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AN OUTBREAK OF UREA POISONING AMONG BANTU FARM LABOURERS IN THE POTGIETERSRUST DISTRICT, TRANSVAAL

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On 7 March 1960 Dr. Ernst Kruger, Superintendent of the General Hospital, Potgietersrust, telephoned and requested my assistance in the investigation of an outbreak of suspected poisoning affecting a large number of Bantu farm labourers. Through the kind assistance of Dr. J. D. Verster, Assistant-Director of Transvaal Hospital Services, and Dr. I. J. Louw, Medical Inspector of Hospital Services in the Transvaal, I undertook the investigation immediately.

HISTORY AND SYMPTOMS OF THE DISEASE

Within 3 - 5 hours after the evening meal the first symptoms, suggestive of violent poisoning, set in, and during the ensuing night some 80 patients were taken to the General Hospital at Potgietersrust. The evening meal consisted, as usual, of cooked meat and mealie meal porridge prepared by a cook for all the Bantu workers in the compound where the affected labourers lived.

The first symptoms were nausea and persistent violent vomiting, followed by extreme excitement and severe general convulsions which resembled those seen in cases of strychnine poisoning. Frequent micturition occurred during the convulsions. Some of the patients were almost moribund when they were admitted to hospital. In spite of the extreme gravity of the symptoms, none of the patients died before I arrived at the hospital early the following morning. Most of the patients were calm, had a slow pulse, and some were asleep, probably as a result of the intravenous administration of chlorpromazine hydrochloride by Dr. J. D. Verster. While I visited the ward where the patients were being treated, one of them again developed a type of convulsion which reminded me of similar convulsions I had seen in sheep and rabbits poisoned experimentally with urea.[1] Dr. Kruger at the same time produced a specimen of Kynoch's Urea Fertiliser which had possibly been mistaken for common salt by the affected patients.

All the patients recovered completely in the course of a few days.

CHEMICAL AND BIOLOGICAL TESTS

A. Chemical Tests

The following specimens were collected: (1) Kynoch's Urea Fertiliser from an opened bag and two specimens from unopened bags, (2) prepared mealie meal porridge, (3) water (from the reservoir) used for cooking the porridge, (4) mealie meal from an opened bag and from the remains in a small bucket, (5) 'Kaffir-beer', (6) vomit, (7) urine, and (8) blood.

None of the well-known convulsant poisons, including urea, was detectable in the specimens of porridge, mealie meal, water, 'Kaffir-beer' and vomit. The specimens of blood and urine showed no significant increase in urea.

The results of the chemical analysis of the 3 specimens of Kynoch's Urea Fertiliser agreed with the details supplied on the manufacturer's containers, namely, 98% urea (= 46% N).

B. Biological Tests

The 3 specimens of Kynoch's Urea Fertiliser were compared with urea B.P. The latter had a 'cooler' and more bitter taste than Kynoch's urea and the bitter taste persisted longer in the mouth than that of Kynoch's Urea Fertiliser.

The toxicity of the 3 specimens of Kynoch's Urea Fertiliser was compared with that of urea B.P. The experiments were conducted upon rabbits and the following facts were established:

1. Kynoch's Urea Fertiliser was approximately 3 times more toxic than urea B.P. The approximate $L.D._{50}$ of Kynoch's Urea was 1·5 G./kg., while that of urea B.P. was 5·0 G./kg.
2. Rabbits, starved for 18 hours, were approximately twice as susceptible to Kynoch's urea and urea B.P. poisoning than unstarved animals.
3. Both Kynoch's urea and urea B.P. are more soluble in 'Kaffir-beer' than in water.
4. Large quantities of the 'Kaffir-beer' or tap water appreciably increased the toxicity of both Kynoch's urea and urea B.P.
5. The symptoms and postmortem appearances induced by Kynoch's urea and urea B.P. were very similar.
6. The fatal dose ($L.D._{50}$) of urea lies very close to that of its toxic dose ($T.D._{50}$), and recovery with toxic, but sublethal, doses occurs very quickly, as is seen in cases of hydrocyanic-acid poisoning.

Symptoms of Urea Poisoning

1. *Urea*

In doses of 15 - 60 G. daily, urea is an effective low-threshold diuretic. It is advisable to take it after meals with water in order to prevent nausea.[2]

According to Sollmann,[3] symptoms of urea poisoning (loss of appetite, nausea, and vomiting) in man appear when the urea nitrogen of the blood is about 70 mg. per 100 ml.

After oral administration of 100·0 G. of urea to man, the blood urea increases to 160 - 245 mg. per 100 ml. in a few hours, and the following symptoms are in evidence: Prostration, apathy, and somnolence.[3] These symptoms are also seen in retention uraemia.

According to Glaister[4], in certain cases of uraemia violent delirium may replace convulsions.

Like many poisons, urea causes disruption of enzymes.[5]

2. *Decomposition Products of Urea*

Under ordinary conditions, urea, administered to man as a diuretic, is excreted unchanged in the urine. However, under certain conditions (see below), urea is decomposed into ammonia (NH_3) and carbon dioxide, especially by the action of urease on it.

Dinnings *et al.*[5] found that ataxia sets in when the blood-ammonia-N level rose above 2·0 mg. per 100 ml. and that death occurred when the ammonia-N increased to 5·0 mg. per 100 ml.

In animals (cattle, sheep, and rabbits) toxic doses of urea induce symptoms typical of ammonia poisoning, namely, irritation of the gastro-intestinal mucosa, restlessness, pronounced excitement, convulsions, and delirium. In fatal cases, death occurs through paralysis of the respiratory centre. If sub-lethal doses of urea have been ingested, or in the case of successful treatment, especially with barbiturates, recovery takes place very quickly. It is the above symptoms in the Bantu labourers which reminded me so much of urea poisoning.

According to the dose of urea administered to the animals, the above symptoms appeared half an hour to 5 hours after administration. Within an hour after administration the urine and faeces of the animals smelt very strongly of ammonia.

Factors which Play a Rôle in the Determination of the Toxicity of Urea

One or more of the following factors may play a rôle in inducing symptoms of ammonia poisoning when excessive quantities of urea are ingested:

(*a*) The presence of some catalyst or other in urea mixtures which assists in the rapid elimination of free ammonia. The 3 specimens of Kynoch's Urea Fertiliser were approximately 3 times as toxic as urea B.P.

(*b*) Starvation renders animals much more susceptible to urea poisoning. It appears that the speed of liberation of ammonia from urea is much quickened by starvation.

(*c*) Large quantities of liquid taken with, or shortly after, urea markedly increase its toxicity. As in the case of starved animals, it appears that the liberation of ammonia from urea is speeded up when it is taken with excessive quantities of liquid. In the above biological experiments, urea dissolved in 'Kaffir-beer' did not appear to be more toxic than when it was dissolved in the same concentration in tap-water.

Urea is most toxic when taken on an empty stomach with large quantities of liquid.

(*d*) Fitzgerald and Murphy[6] studied the distribution of urease in the tissues of different species and found that it is widely distributed in all animal species. The concentration of urease is far higher in the stomach than in the duodenum. The amount of urease in the gastric mucosa is variable and can be increased by (1) a high protein diet, (2) a soybean diet, (3) injection of urogastrone, and (4) a high urea intake. It is interesting to note that enterogastrone had no effect on the concentration of urease in the gastric mucosa.

(*e*) Bernheim and Bernheim[7] found that 'caffeine injected into rabbits causes a slight diuresis, no change of the pH of the urine, an increase in the NH_3-N excretion, and some decrease in the urea-N excretion'. The longer urea is retained in the gastro-intestinal tract, the greater the likelihood of its being split into ammonia and carbon dioxide.

(*f*) Experimenting on male West Africans and comparing them with European men, Kenney[8] found that, when urea is administered to these males, the West Africans excrete less of it than the European men. This may possibly result in more urea being broken up into ammonia and carbon dioxide in the African than in the European.

TREATMENT

Treatment is symptomatic. It appears that tranquillizers (e.g. chlorpromazine) serve a very useful purpose in quietening the delirium and convulsions. The intravenous administration of suitable barbiturates would also have a beneficial effect. In moribund patients, where the violent delirium and convulsions are followed by coma and beneficial effect. In moribund patients, where the violent should be wisely used. The less active analeptics ('megimide', methedrine, amiphenazole) should be tried first since the more active ones (picrotoxin), in large doses, are more inclined to cause dangerous secondary depression of the vital centres in the cortex.

DISCUSSION

There seems little doubt that the serious outbreak of disease among the Bantu farm labourers was caused by the fact that they had mistaken a bag of Kynoch's Urea Fertiliser for common salt. Unfortunately, the common salt was stored in the same room as the urea fertiliser. According to Sollmann,[2] 100·0 G. of urea induce symptoms of poisoning in man. It would therefore seem very unlikely that an adult could consume this quantity of urea (mistaken for common salt) in the course of one meal. However, the following factors played a very important rôle in the outbreak of urea poisoning under consideration: (*a*) The Bantu labourers had not had common salt for quite a period and were thus inclined to consume larger amounts with their food than they would ordinarily have done; (*b*) the labourers did manual work in a hot and dry climate, consequently, their salt intake would be appreciably higher than in people not exposed to such hard physical exercise; (*c*) it is customary among many Bantu peoples not to eat on Sundays until the late afternoon or evening, as happened in the above case; consequently they consumed a large quantity of porridge which, in turn, resulted in the consumption of large quantities of salt, and the starvation made them more susceptible to urea poisoning; (*d*) further, it is a general custom among these people not to mix common salt into their food, but to place it in the hollow of their one hand, or in the lid of a tin, and dip every bit of meat or porridge in the salt. In this way, I have seen them ingesting an ounce, or more, of common salt in the course of one meal; (*e*) during week-ends, and especially on Sundays, Bantu people consume large quantities of 'Kaffir-beer', and there was ample evidence that the labourers concerned in this case had consumed large quantities of beer with the evening meal which preceded the outbreak of the disease.

SUMMARY

An outbreak of urea poisoning in Bantu farm labourers as a result of mistaking Kynoch's Urea Fertiliser for common salt is described.

I wish to thank Dr. Ernst Kruger, Superintendent of the Potgietersrust Hospital, and Dr. J. D. Verster, for details concerning the history, symptoms, and treatment of the disease; Dr. L. S. de Villiers, Senior Lecturer, Department of Pathological Anatomy, University of Pretoria, and members of his staff for the chemical analysis of the food, blood, and urine specimens collected; Dr. Tom Adelaar and Mr. J. A. Minnie of the Section of Toxicology, Onderstepoort Laboratories; and Dr. F. W. K. Grotepas, Senior Lecturer, Department of Physiology, University of Pretoria, for the chemical analysis of the specimens of Kynoch's Urea Fertiliser.

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VAN DIE REDAKSIE : EDITORIAL

DIE MEDIESE LEERPLAN

Een van die belangrike en moeilike probleme waarvoor die opleidingshospitale vandag te staan gekom het, is die probleem van hoe om die leerplan aan te pas by die versnelde tempo en wydlopende uitbreiding van die moderne mediese kennis. Hierdie probleem is natuurlik nie net eie aan die mediese opleidingsinrigtings nie. In byna alle vertakkinge van die wetenskap en van akademiese aktiwiteit in die algemeen, is daar die neiging tot 'n geweldige uitbreiding van kennis. En orals het daar die tendens ontstaan vir spesialiste en superspesialiste om te ontwikkel.

In die letterkunde, veral wat betref akademiese kritiesliterêre werk, neem hierdie neiging die vorm aan wat ons graag wil noem stiletstudies — delwing wat dieper en dieper gaan oor 'n kleiner en kleiner gebied, bv. doktorsverhandelinge oor 'n frase, of 'n woord, of selfs die weglating van woorde! In die natuur- en toegepaste wetenskappe word die beweegruimte wat navorsers hulself toelaat ook al meer omskrewe en afgemeet. En in die medisyne vind iets soortgelyks plaas. In die optalmologie alleen is daar bv. 'n groot aantal super-spesialiste, elkeen 'n deskundige op 'n onderafdeling van die optalmologie. Daar is spesialiste in aspekte van hartsiekte, diabetes, endokrinologie, ens.

Ons het daar geen beswaar teen dat persone onderafdelings van hierdie onderwerpe en vraagstukke bestudeer nie. Trouens, dit is hoe ons kennis groei. Maar, dit lyk nie reg dat so 'n groot aantal mense hul hele bruikbaarheid oor 'n leeftyd beperk tot 'n onderafdeling van 'n vak nie. Êrens gaan die geneesheer verlore in die ontstaan van die tegniese meester. En ons eie gees en die van ons pasiënte is daar swakker aan toe — ook miskien ons algemene gesondheid, en bepaald ons geestesgesondheid. Nie net diepte van spesifieke kennis nie, maar ook omvangrykheid van perspektief kenmerk 'n werklike groot mens.

Teen hierdie algemene agtergrond moet die inkleding van die mediese leerplan van die toekoms dus gesien word. Die onderwerp is natuurlik glad te groot om hier uitvoerig en bevredigend behandel te word. Nogtans wil ons 'n paar gedagtes uitspreek wat mag bydra tot die rigting waarin die gedagtes van die beplanners van ons opleiding mag beweeg.

In die verlede is die maklikste oplossing heel dikwels gevolg, naamlik, al wat nuut was of nuut bygekom het, is maar net steeds by die bestaande leerplan gevoeg. Die gevolg is dat ons al meer die gevaar loop om 'n oorlaaide leerplan en oorstuurde dosente en studente te kry. En nou het ons werklik die punt bereik waar daar nie meer bygelas kan word nie.

Wat ons dus te doen staan is nie net heroorweging en herindeling van die leerplan nie. Daar moet 'n basiese heroriëntasie kom met betrekking tot wat ons *wil* met mediese opleiding en ook met betrekking tot wat ons *kan*.

In die eerste plaas sal opleiding in terme van algemene beginsels weer 'n relatiewe groter plek moet inneem in teenstelling met detailopleiding. Ons kan die detail-stryd op die duur nie volhou nie. En as ons sou probeer om dit te doen, sal ons tog maar die aftog moet blaas.

In Suid-Afrika met sy groot binnelandse afstande en wydverspreide bevolking, sal die algemene praktisyn (wat goed toegerus is met kennis van uiteenlopende aard en met gesonde verstand wat berus op 'n breë perspektief) nog oor baie toekomsjare die sleutelfiguur bly in ons mediese dienste. Ook sal ons by die heroriëntasie waarvan ons gepraat het, meer aandag moet gee aan die breë kulturele ontwikkeling van ons studente. Hand aan hand met die vinnige uitbreiding van feitekennis op al die gebiede waarna ons verwys het (en al die baie ander waarna ons nie verwys het nie), gaan die neiging op byna alle gebiede om geleerde tegnici te kweek. (Die term geleerde barbare is te sterk, maar daar is nogtans 'n versoeking om dit te gebruik.) Hierdie gevaar staar ons orals aan — in die regte, in die ingenieurswese, in die natuurwetenskappe, en in die medisyne.

Die probleem waarvoor ons as beskaafde wêreld staan, so wil dit voorkom, is nie meer om 'n man op die maan te laat land nie. Dit sal sonder twyfel eersdaags geskied, en natuurlik nie sonder die bykomstige trompetgeskal wat deesdae so kenmerkend is van ons ondersonse (want die maan is al te naby) tranedal nie. Die probleem, sê ons, is nie meer om 'n man op die maan te laat land nie, maar om hom hier te hou en hom in staat te stel om in vrede en welwillendheid met sy ewenaaste saam te leef in 'n lewe wat vir hom en vir hulle ryk is aan skeppingsvreugde en arbeidsgenot.

Om hierdie doel te bereik is die uitdaging waarvoor al ons akademici dwarsoor die wêreld staan. Laat ons ook in die medisyne en in die opleiding van ons geneeshere en heelmeesters van die toekoms hierdie uitdaging aanvaar in die wete dat dit nog nooit *net* die kennis was wat die wêreld verlos het nie, maar *ook* die liefde.

THE BLOOD-BRAIN BARRIER

The observation by Ehrlich in 1885 that certain aniline dyes injected into the blood stream stain all tissues of the body except the central nervous system, and subsequently noticed for many other substances, gave rise to the idea that there is a blood-brain barrier (BBB). Most workers have moved away from the concept that there is a structural barrier, towards the idea of a mechanism or system of blood-brain relationships involving dialysis, ultrafiltration, osmosis, the Donnan equilibrium, electrical changes, lipoid solubility, special tissue affinity, or metabolic activity.[1] With exact knowledge it would be unnecessary to use the term barrier. Many still believe that the term BBB is a special property of the brain capillary endothelium or some related membranous structure. Some believe the barrier has a protective function, preventing access to the brain of toxins, bacteria, toxic dyes, excess of electrolytes or colloids, and metabolites, and promoting a brain homeostasis. Others conclude that the metabolism

of the brain cells governs the function of the barrier, much depending on the exclusion or assimilation by the cerebral cells themselves. There is thus the possibility that there is no morphological evidence of a structural barrier, and that distinctions between the brain's behaviour and that of other organs may not be correct. Most of the dyestuff evidence is now discredited, so that the widespread assumption from such studies — that there is a structural BBB — falls away, and reconsideration of the subject on the basis of physiological principles is now needed. The BBB must be considered as a reflection of *in vivo* cerebral metabolism.

The pharmacology of the central nervous system is an enormous subject that is expanding very rapidly. BBB concepts repeatedly arise with regard to the access of particular drugs to the brain and in relation to normal metabolism. It has sometimes been more convenient to study blood — cerebrospinal-fluid relationships, involving the rate of transfer of one to the other, and the ratio at equilibrium. There is much difficulty in such studies in the methods used, the techniques of assay, and the interpretation. Failure to produce pharmacological actions on the central nervous system may not mean that a drug has not entered the system, especially when it is known to be rapidly metabolized. Again, an apparent central action may arise from effects produced, for example, on the carotid sinus or on blood carbon-dioxide tension, i.e. from outside the central nervous system. Metabolic explanations may eventually be found for the apparent barrier to many drugs. The consideration of cerebral metabolism as a factor in the penetration of drugs to the brain has hardly begun.[1]

The finding that permeability of the barrier is reduced in pathological states has led to the idea that increased permeability may be a primary causative factor in the causation of certain diseases of the central nervous system. Thus immaturity of the barrier is regarded as permitting kernicterus, but all the facts are explained if anoxaemia is accepted as the primary factor in the pathogenesis of this phenomenon. It is being suggested at the present time that other conditions, such as epilepsy and disseminated sclerosis, may be the result of the breakdown in protection normally afforded by the BBB; but it is more likely that the penetration of substances into nervous tissue may be the non-specific result of unknown primary pathology rather than a primary factor in their pathogenesis.

Evidence of the BBB has been based by many workers on the comparison of the ability of nervous tissue with that of other tissues to take up substances from the blood. It is necessary, however, first to demonstrate that entry is not restricted by the tissue's relative inability to metabolize the substance concerned at a sufficient rate. The brain, with its own highly specialized functions, differs metabolically from the liver, for example, which metabolizes over a wide range on behalf of the whole body. It is therefore unwise to attribute comparatively slow rates of entry into the brain to a BBB on such evidence alone. The plea is made that, until the rate of entry of a metabolite into the brain can be demonstrated to be a restriction on its utilization, it should be assumed to be a reflection of, rather than a limiting factor in, *in vivo* cerebral metabolism.[1,2]

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FAMILIAL NEPHROPATHY WITH SPLENOMEGALY

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The existence of hereditary nephropathy was recognized in the 19th century, and in 1930 Mitchell[1] was able to review no less than 33 papers dealing with this subject since 1874. More recently, a number of reports have appeared describing genetically determined renal disease, with manifestations ranging from symptomless proteinuria and microscopic haematuria to uraemia, hypertension, or the nephrotic syndrome. A remarkable feature of many of these has been the coexistence of other defects, especially nerve deafness and abnormalities of the eye, such as spherophakia and cataract. In general, males appear to have been more frequently and more severely affected than females. It should be stated that certain forms of renal tubular disease, such as the Fanconi syndrome and renal tubular acidosis, which may also be genetically determined, are not under consideration here.

Perkoff *et al.*[2] were able to trace 134 of 232 direct descendants of a single person, and found that no less than 50 of them suffered from proteinuria, pyuria and hypertension. In this family there were 7 cases of fatal uraemia (all in males), 8 of progressive nerve deafness, and 19 of mitral-valve disease. Autopsies performed on 2 patients revealed pyelonephritis. Reyersbach and Butler[3] reported 8 patients with congenital haematuria, 3 of whom had nerve deafness, in 4 unrelated families. Renal biopsy in 1 patient showed no abnormality other than red cells in the tubules. Many cases of nerve deafness, eye defects, and nephritis were present in the relatives of one of these groups.

Sohar[4] described an Israeli family in which 4 brothers and a sister had proteinuria with erythrocytes, leucocytes and granular casts in the urine. All the brothers had elevated blood-urea levels, decreased urine-concentrating power and urea clearances, and nerve deafness. In addition, 2 had slight hypertension, 2 spherophakia and 2 congenital posterior cataracts. The mother and 2 maternal uncles also suffered from nerve deafness and kidney disease. Goldbloom *et al.*[5] reported a mother and 3 sons of Jewish stock who had renal disease. Two of the sons had nerve deafness and one of these had bilateral anterior sub-capsular cataract. Postmortem examinations on the 2 sons who died showed severe pyelonephritis.

Vernier *et al.*[6] presented the results of a study of a family in which all 4 children suffered from either 'pure' nephrosis or chronic glomerulonephritis with the nephrotic syndrome. The presence of nephropathy was anticipated in the fourth of these children, and the first urine voided after birth was shown to contain protein and a few red

and white cells. Biopsies of the kidneys were done in all 4 patients and these revealed changes ranging from the normal to advanced glomerulonephritis. Wallace and Jones[7] described a family in which 3 children died of chronic renal disease and hypertension; autopsies were carried out on 2 of the children, and chronic glomerulonephritis was found. There was increased amino-aciduria in 1 of these as well as in 2 surviving siblings and in both parents.

It is our purpose to present the details of a Jewish family consisting of a father and 3 sons who suffered from apparently benign proteinuria. A renal biopsy was carried out in one of the sons; this showed histological abnormalities of the glomeruli.

CASE REPORT

L.W., the father, aged 57, had been found to have proteinuria at a routine insurance examination 8 years previously. Five years later a papilloma of his bladder was fulgurated and this operation was complicated by an air embolus resulting in partial hemiplegia. S.W., P.W., and H.W., his sons aged 27, 24, and 21 respectively, were also found, on routine examination, to have proteinuria. This discovery led to a more detailed study.

Abnormalities of the urine were not present in 2 of the father's sisters nor in his brother. Findings in a third sister were unobtainable. His wife's urine was normal. Neither L.W. nor his 3 sons had suffered from renal or other disease except for exanthemata of childhood including scarlet fever. Apart from slight enlargement of the spleen in S.W. and H.W., the physical examinations were normal and the blood pressures were not raised. The proteinuria, which was present in recumbency in each instance, increased on standing. The amount of protein excreted in 2 24-hour periods is shown in Table I. Glycosuria was not present and bacterial cultures of the urine were negative. Microscopic examination of the urine showed increased numbers of red cells, white cells, and casts. The casts were mainly hyaline and granular, but occasional doubly refractile casts were also seen. Addis counts confirmed the considerable increase in formed elements, especially red cells (Table II). Amino-acid chromatography of the urine showed that amino acids were present in normal amounts and no abnormal amino acids were found.

TABLE I. URINARY PROTEINS

		L.W.	*S.W.*	*P.W.*	*H.W.*
Protein excretion* (mg. per 24 hours)		348	388	356	823
		432	204	263	—
Mucoproteins		Faint trace	Faint trace	Faint trace	Faint trace

* In all 4 cases albumin accounted for 70 - 80 per cent of the urinary protein excretion.

TABLE II. ADDIS COUNTS (TWO 24-HOUR SPECIMENS)

		L.W.	*S.W.*	*P.W.*	*H.W.*
Leucocytes (millions)	1st	1·74	3·90	4·40	3·81
	2nd	0·92	0·92	9·40	—
Erythrocytes (millions)	1st	41·60	69·60	724·68	55·40
	2nd	25·34	1·73	2,001·62	—
Casts (thousands)	1st	1,740	173	2,820	952
	2nd	9,620	188	5,040	—

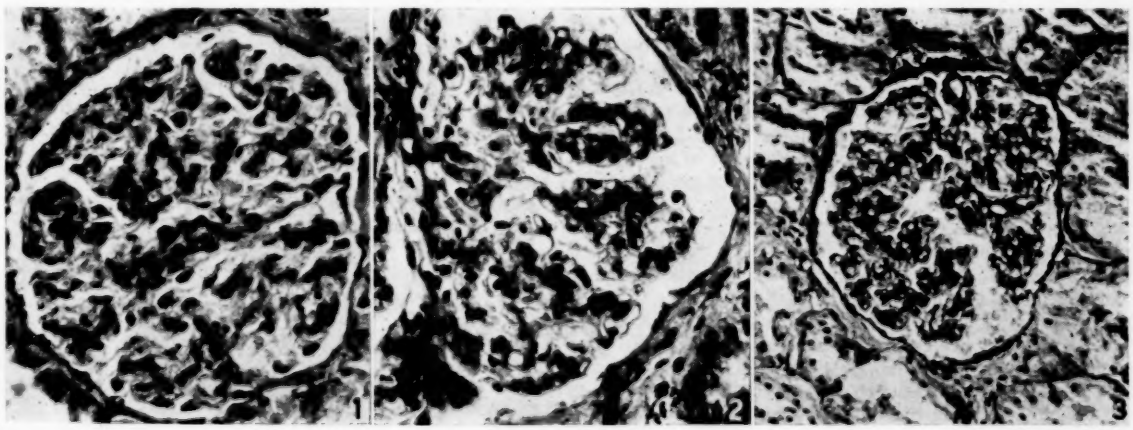
Standard concentration and dilution tests of the urine were carried out on L.W., S.W., and P.W., but not on H.W., who had gone abroad by the time this study was undertaken. The urine could be concentrated to 1019, 1021 and 1023 and diluted to 1002, 1003 and 1001 respectively. The corresponding urea-clearance values were 86%, 103% and 95% of normal. Results of the phenolsulphothalein-excretion tests were normal.

The following investigations were also normal in all 4 patients: haemoglobin, packed-cell volume, white-cell and differential counts, erythrocyte sedimentation rate, prothrombin index, serum urea, potassium, sodium, chloride, bicarbonate, cholesterol, calcium, inorganic phosphorus, alkaline phosphatase and uric acid. The serum-protein electrophoretic patterns were normal and screening tests for syphilis were negative. The urinary electrophoretic pattern showed 70 - 80% of albumin in each case (Table I). Malrotation of the kidneys was demonstrated on intravenous urography in L.W. and P.W., but this examination was otherwise normal in all 4 patients.

Percutaneous renal biopsy was performed on H.W., chosen because his proteinuria was most marked. The glomeruli proved to be larger and more cellular than normal, and contained excessive numbers of polymorphonuclear leucocytes as well as a few macrophages (Fig. 1). There was separation of the loops into quite distinct tufts (Fig. 2) with adhesion of some of these to the capsule. Several small areas of hyalinization were also present in the glomeruli. Periodic-acid Schiff preparations revealed thickening of the basal membrane (Fig. 3). The tubules were normal. These findings were regarded as compatible with glomerulitis, such as is seen in the recovery stage of acute glomerulonephritis.

DISCUSSION

A distinction must be drawn between those familial nephropathies which are the result of a common environmental factor, such as streptococcal infection or a toxin, and those which are inherited. This may not be easy when all the patients occupy the same house and their relatives



Figs. 1 - 3. See text.

are unaffected. Although first discovered in adult life, there is no reason to believe that the renal disease in L.W. and his 3 sons was caused by external agents and it is considered more likely to be of hereditary origin.

Because of the lack of significant impairment of renal function in the father at the age of 57, the disease may be expected to follow an equally benign course in the sons. A further reason for expecting this is the relatively mild degree of pathological change demonstrable at renal biopsy in the son with the most marked proteinuria. On the other hand, the possibility that renal insufficiency will ensue in the course of time cannot be completely excluded.

The splenomegaly present in S.W. and H.W. is a feature which has not previously been noted in association with hereditary nephropathy, but nevertheless it does not seem likely that it is merely coincidental. The true significance of this unusual finding remains a matter for speculation.

It is interesting to note that renal disease, especially pyelonephritis, has recently been described in combination with Marfan's syndrome[5] which may also, in some cases at least, be a genetically determined condition.[9] However, evidence of Marfan's syndrome was not present in our patients, nor was it mentioned in any of the other reports. Another aspect which could be relevant from the genetic view-point is the relatively high proportion of Jewish families among those suffering from hereditary renal disease, viz. the families reported by Sohar[4] and Goldbloom *et al.*,[5] and the one we describe. Whether this represents a real racial susceptibility is uncertain.

On reviewing the histological appearances of the kidneys in those examples of hereditary nephropathy referred to, it is possible to classify them into cases of pyelonephritis and into those which seem to conform to the diagnosis of glomerulonephritis in its various phases. If this interpretation is correct, all these cases of hereditary renal disease fall into two categories despite their widely differing clinical presentations.

It is even possible that the relationship between these two is closer than is apparent, in view of the occurrence of eighth-nerve and eye disorders in both. This 'unitarian concept', as far as the various forms of glomerulonephritis are concerned, is supported by Vernier *et al.*[6] In the family he studied, the clinical manifestations included transient 'pure' nephrosis, persistent 'pure' nephrosis, so-called mixed nephrosis-nephritis, and chronic glomerulonephritis. At biopsy, the corresponding histological features ranged from the normal to severe chronic glomerulonephritis.

SUMMARY

A family is described in which the father and his 3 sons were found to have symptomless proteinuria.

Unexplained splenomegaly was present in 2 of the sons.

Addis counts revealed an increase in formed elements, especially red cells, in all 4 patients.

A renal biopsy was performed in the most markedly affected of the 4; this showed a mild glomerular lesion suggestive of glomerulonephritis.

In all probability the nephropathy in this family is hereditary in origin.

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TOXOPLASMOSIS

A REPORT OF FOUR CASES

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To date only 5 cases of toxoplasmosis have been reported from this country, and of these 3 were diagnosed postmortem. Klenerman[1] recorded the first case in 1951—an African child presenting with an encephalitic picture at the age of 6 weeks and dying after 2 days, in whom the diagnosis was made on autopsy material. The mother's serology provided confirmatory evidence. The second case, that of a 4-year-old European child with mental retardation, choroido-retinitis and cerebral calcification, was reported by Rabkin and Javett in 1952.[2] Becker, in 1954,[3] recorded 2 cases diagnosed postmortem—an infant of 5 months with hydrocephalus, and a child of 4½ years who presented a picture of acute encephalitis and died after a 6 weeks' illness. Fasser, in 1955,[4] reported the case of a 2-year-old Indian child with mental retardation, hydrocephalus, and cerebral atrophy, but with no retinal changes or cerebral calcification.

The purpose of this paper is to report a further 4 cases, all diagnosed during life, and all in European children in Durban. It is not our intention to review the subject, for this has recently been done in this *Journal* by Spencer;[5] and other excellent reviews, such as that by Feldman,[6] are available to the interested reader.

It is not out of place, however, to recall that the parasite, a protozoon, first described in 1909 by Nicolle and Manceaux[7] in the North African rodent, *Ctenodactylus gondii*, is widely distributed geographically and has an unusual lack of host specificity, infection having been reported in a great variety of vertebrates. Human infection, apart from the congenital form, is presumably acquired accidentally from contact with animals. Since 1939, when the first human cases were described,[8,9] it has become evident from serological surveys that toxoplasmosis is a common infection in man. The vast majority of acquired infections, however, are subclinical, giving rise to no recognizable disease. Conventional classification groups the manifestations of acquired disease into glandular, ocular, encephalitic, and exanthematous forms.

Of the 4 cases presented here, the first 2 illustrate the

congenital type of toxoplasmosis, case 3 the ocular type, and case 4, reported in some detail, shows widespread systemic involvement with the production of a most confusing clinical picture.

CASE REPORTS

Case 1

L.G. was the fourth child of normal, healthy parents. Pregnancy had been perfectly normal and labour was uncomplicated. The birth weight was 7 lb. 11 oz. The 3 elder siblings were well.

She presented first on 18 March 1954, at the age of 4½ months, with a history of feeding difficulty since birth, and generalized convulsions with loss of consciousness. The mother had observed 16 convulsions during the previous 3 weeks.

Examination revealed a well-nourished child with a head circumference of only 14½ inches, who was obviously grossly retarded. Apart from some questionable pallor of the discs, the fundi were normal. X-ray of the skull revealed paraventricular and tentorial calcification. Subsequent pneumo-encephalography showed 'dilatation of the falcine and tentorial spaces and of the cisternae, and ? ventricular dilatation. The hemispheres appear retracted'. The cerebrospinal fluid (CSF) showed no abnormality. Serum calcium was 12·0 mg. per 100 ml., alkaline phosphatase 17 K-A units, and WR negative.

Serological tests for toxoplasmosis were carried out in Hanover, Germany, and were reported as follows: Sabin-Feldman test, positive 1 : 64; complement-fixation test, uncertain negative; and cadmium-sulphate test, +++. The maternal serum unfortunately became contaminated.

The child was seen repeatedly until the age of 1 year, when admission to a mental institution was arranged. She made virtually no progress and the fits were not controlled by a variety of anticonvulsants.

Case 2

M.W. presented on 30 January 1956, at the age of 4 months, with a history of diarrhoea for 2 months, vomiting for 3 weeks, and fever and irritability for 12 hours. He was the only child of healthy parents and the product of a normal pregnancy and delivery. Birth weight was 8 lb. 12 oz.

Examination revealed an extremely irritable baby with a temperature of 102° F.; the eyes were constantly deviated to the left. There was no neck stiffness, but the fontanelle was slightly tense. Lumbar puncture produced clear CSF containing 3 lymphocytes per c.mm. and protein 70 mg. per 100 ml., with a moderate increase of globulin.

The following day he developed a left sixth-nerve palsy, and the fever continued for 9 days, during which time he was drowsy and irritable when disturbed. Examination of the fundi on 2 February showed pigmented areas of choroido-retinitis in both eyes, typical of toxoplasmosis. The blood count was normal. Head X-ray revealed no intracranial calcification or other pathology. He was treated with sulphadiazine, and when he was discharged on 22 February the strabismus had disappeared and he seemed well. The CSF at this time contained 1 lymphocyte per c.mm. and protein 35 mg. per 100 ml., with only a slight increase of globulin. A month later he was apparently well and thereafter could not be traced. Both the patient's and his mother's sera were sent for dye and complement-fixation tests, but the records of these results were lost. The clinical picture in itself, however, leaves little doubt that this child was suffering from toxoplasmosis.

Case 3

L.B., a girl of 12 years, was admitted in August 1959 for investigation of deteriorating vision. This had first been complained of in 1957 when a choroido-retinitis of the right macular region was discovered. Since then the lesion had progressed until she had no central vision in the right eye. Recently similar changes had been observed in the left eye though vision was, as yet, unimpaired.

Her past history was complex, including 3 episodes of 'glandular fever' in 1951, 1953 and 1954, each time, it was said, with a positive Paul-Bunnell test. In 1952 she had a febrile illness and was investigated for, *inter alia*, tuberculosis and leukaemia. In 1956 she was treated for alleged amoebic hepatitis. In January 1958 she was said to have had tick-bite fever, and up to the time of admission was getting recurrent episodes of fever with glandular swellings.

Examination was essentially negative apart from the eyes. The right macular region was destroyed by a patch of choroido-retinitis which appeared inactive. Similar, but much less marked, changes were present in the left eye. A blood count showed no abnormality, and the ESR was 6 mm. per hour (Wintrobe). The Paul-Bunnell test was negative. Head and chest X-rays revealed no abnormality. The CSF was normal.

The whole history was so suggestive of toxoplasmosis that we were surprised and disappointed when the tests proved negative. However, the child was subsequently seen and re-investigated in Johannesburg when the Sabin-Feldman dye test was positive (1 : 64) and the toxoplasma complement-fixation test positive at 1 : 4 and dubious at 1 : 8. Opinion differed among the 8 ophthalmologists who saw her regarding the activity of the lesion, but she was treated with methylprednisolone, sulphadimidine and pyrimethamine, and to our knowledge there has been no further progression of the disease.

Case 4

F.M., a boy of 11 years, was admitted in July 1958, with a 4-day history of fever, headache, pain in the neck, lethargy, vomiting, constipation, photophobia and a dry cough. Previous illnesses included pertussis, measles and rubella. Later a history of haematuria 4 years before admission was obtained, and he admitted to frequent exposure to bilharzia in the Umhlatuzana River, a notorious local source of both *S. haematobium* and *S. mansoni*.

Examination revealed a small, ill boy, febrile (Fig. 1), with slight non-tender cervical lymphadenopathy, a liver enlarged 2 inches below the costal margin, and a very large, firm, tender spleen extending to below the level of the umbilicus. There were no signs of meningitis, and the fundi were normal on admission. He presented, therefore, as a patient with pyrexia of unknown origin associated with hepatosplenomegaly.

Special investigations were performed as follows:

1. *Blood count.* Haemoglobin 10·0 G. per 100 ml., PCV 29%, MCHC 34%, ESR 28 mm. per hr. (Wintrobe), WBC 4,000 per c.mm., neutrophils 40%, lymphocytes 54%, eosinophils 5%, basophils 1%. Repeated counts failed to demonstrate an absolute eosinophilia of more than 440 per c.mm. Red-cell fragility was normal. LE cells were not found.

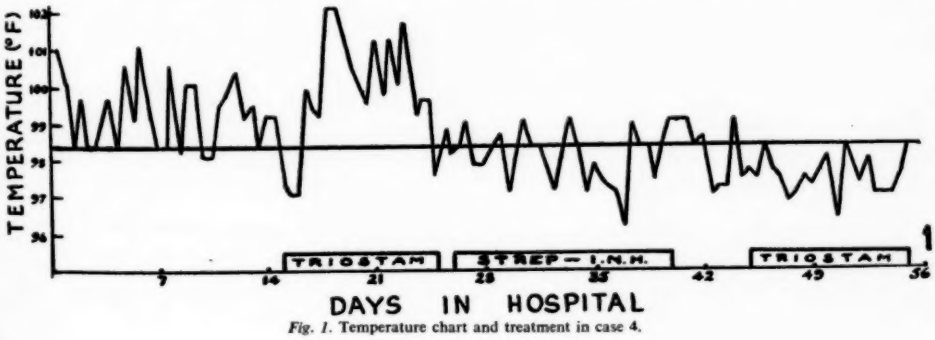

Fig. 1. Temperature chart and treatment in case 4.

2. *Urine examination.* Repeated examinations, including 24-hour specimens, failed to reveal any ova.

3. *Stool examination.* Repeated examinations showed trichuris ova only. No schistosomal ova were demonstrated.

4. *X-ray studies.* Chest: 31 July—'No evidence of any pathology noted'. 6 August—'right upper and mid-zone inter-

stitial consolidation. Remainder of the lung fields show generalized plethora with appearances suggestive of alveolar involvement. There is hilar calcification'. 21 August—'Perihilar adenopathy. There is bilateral, diffuse, increasingly confluent lung mottling with pulmonary hyperaemia—? reticulosis, sarcoidosis or lymphoma'. No pathology was demonstrated in the skeleton.

5. *Agglutination tests.* All tests for typhoid, paratyphoid, typhus, and brucellosis were negative. The Paul-Bunnell test was also negative.

6. *Tuberculin tests.* Patch and intradermal tests were negative. Gastric washings were negative for acid-fast bacilli.

7. *Liver-function tests:*

	5 August	*14 August*	*24 October*
Van den Bergh reaction ..	neg.	neg.	neg.
Bilirubin (mg. per 100 ml.) ..	0·7	0·5	1·0
Alkaline phosphatase (K-A units)	4·0	40·0	18
Zinc turbidity (units)	18	16	10
Cephalin cholesterol	4	4	1
Thymol turbidity (units) ..	9	10	7
Total protein (G. per 100 ml.) ..	9·2	8·8	8·8
albumin (G. per 100 ml.) ..	3·7	3·7	4·7
globulin (G. per 100 ml.) ..	5·5	5·1	4·1

8. *Bone-marrow examination.* This showed a slight leucocytosis with a mild eosinophilia only.

9. *Rectal biopsy* done on 11 August demonstrated old degenerate ova of *S. haematobium*, but *S. mansoni* ova were not found.

10. *Liver biopsy* on 27 August. Serial sections showed focal granulomata with an eosinophilic cellular reaction suggestive of bilharzia, but no ova were seen on differential staining. A mild periportal fibrosis was also present.

11. *Splenic biopsy* also performed on 27 August merely showed increased fibrosis and pigment deposits. No ova were found.

(Subsequent re-examination revealed no toxoplasma in either liver or spleen biopsy specimens.)

After 2 weeks in hospital the boy's general condition had deteriorated considerably. He was still pyrexial and cough was a troublesome symptom. The only positive findings were the pulmonary radiological changes and the schistosomal ova on rectal biopsy. In spite of the low eosinophilia it was decided to treat him for bilharzia, and he was accordingly given a course of antimonyl gluconate ('triostam'), 75 mg. daily for 10 days. In spite of the unusual febrile response (Fig. 1) he improved remarkably towards the end of the treatment.

Three weeks after admission fundoscopy was repeated, and lesions were seen consisting of small yellow patches, one in each eye, located about 2 disc-diameters above the optic discs. These were so suggestive of tubercles that the patient was started on a course of streptomycin and INH, but this was stopped when it was felt that his clinical condition was incompatible with a diagnosis of miliary tuberculosis. The ocular lesions progressed, gradually hardening in appearance, until at the time of discharge from hospital they appeared as white areas of choroido-retinitis with a thin rim of pigment, indistinguishable from healed tubercles. They have remained unchanged.

Although the ocular lesions should have suggested a diagnosis of toxoplasmosis, they were regarded as possibly bilharzial granulomata, and a second course of triostam was administered. The patient was then well, but the enlargement of the liver and spleen persisted, receding slowly over the following year. Only in May 1959 was the patient investigated for toxoplasmosis. The dye test proved positive to a titre of 1 : 1024, and the complement-fixation test was positive 1 : 4. Since there was then no evidence of activity of the disease he was given no further treatment.

DISCUSSION

Cases 1 and 2 illustrate the congenital form of toxoplasmosis in which the infection is acquired by the foetus in utero as a result of (usually clinically inapparent) maternal infection with parasitaemia. This type may present various manifestations, among which choroido-retinitis and cerebral calcification are common. Other features include hepatomegaly, splenomegaly, jaundice, skin rashes, hydrocephalus, microcephalus, myocarditis, and still-birth. Later, convulsions and mental retardation are found in the vast majority of survivors.

Case 3 illustrates the common glandular manifestations of acquired disease. Glandular fever may be simulated, not only clinically, but also in regard to the blood picture. The Paul-Bunnell test is, however, alleged to be always negative in toxoplasmosis. In this child it was said to have been positive on several occasions before we saw her, but was completely negative when done in hospital. In addition to the febrile lymphadenopathy this case demonstrated typical toxoplasmic uveitis, which may occur as a very late manifestation of acquired infection.

Case 4 presented with extensive visceral involvement, manifested by marked hepatosplenomegaly, pulmonary infiltration and fever. The subsequent choroido-retinitis should, perhaps, have led to an earlier diagnosis. Of considerable interest in this case was the apparent favourable response to treatment with antimony. This has not, to our knowledge, been previously recorded, and it would appear to be a line of therapy worthy of further investigation. The results of standard treatment—sulphadiazine and pyrimethamine—are generally satisfactory in the acquired case, in that the disease is halted and further damage prevented. In the congenital form, however, there is usually extensive and irreversible destruction of cerebral tissue and treatment is, therefore, of little avail.

SUMMARY

Four cases of toxoplasmosis in European children—2 congenital and 2 acquired—are reported, all of which were diagnosed during life.

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IDIOPATHIC CARDIOMYOPATHY SIMULATING ORGANIC VALVE DISEASE

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Cardiomyopathy of unknown origin is extremely common in the African population of Johannesburg.[1-4] As in the cardiomyopathies in general,[5] the heart valves are normal and the principal auscultatory findings are gallop rhythm and functional systolic murmurs caused by dilatation of the mitral and tricuspid rings. Diastolic murmurs are generally absent.

The object of this paper is to describe 2 cases of idiopathic cardiomyopathy which presented with both diastolic and systolic murmurs of functional origin.

These cases simulated organic disease of the mitral and aortic valves, owing to the persistence of the murmurs and the peripheral circulatory findings.

CASE REPORTS

Case 1

E.S., an African male aged 13 years, was admitted to hospital in July 1959, complaining of swelling of the lower limbs and a dry cough of 2 weeks' duration. He had been having bouts of palpitations associated with dizziness for 'a long time' and, on questioning, admitted to breathlessness on exertion and episodes of paroxysmal nocturnal dyspnoea. There was no history of rheumatic fever.

On examination the patient was orthopnoeic and in severe congestive cardiac failure. He was apyrexial with a regular pulse rate of 100 per minute and a blood pressure of 110/60 mm.Hg. The pulse volume was poor and the extremities were cool. There was pronounced anterior bulging of the praecordium. The heart was markedly enlarged, with heaving impulses over both ventricles. A systolic thrill was palpable at the base and radiated into the neck. A systolic thrill was also felt at the apex. Auscultation at the apex revealed a protodiastolic gallop and a loud pansystolic murmur radiating into the axilla and around to the back. At the aortic and pulmonary areas there was a harsh mid-systolic murmur radiating into the neck. At the aortic area and down the left sternal border an early diastolic blowing murmur was audible. The pulmonary second sound was accentuated and closely split.

Radiography confirmed the gross cardiomegaly, with both left and right ventricular enlargement. The aortic knuckle was rather small and the pulmonary outflow tract was prominent. Posterior displacement of the barium-filled oesophagus and widening of the carina suggested left atrial enlargement. Electrocardiography showed left ventricular hypertrophy and strain (S in V_2 + R in V_6 = 50 mm.; T in V_6 inverted). The haemoglobin level was 13·9 G. per 100 ml., the ESR 5 mm. in the first hour (Wintrobe), and the leukocyte count and urine examination were normal.

The diagnosis made was that of congestive cardiac failure caused by aortic stenosis and incompetence and mitral incompetence, probably rheumatic in origin.

Following the administration of digitalis and chlorothiazide, the patient's condition improved slightly, but on the 4th day after admission he suddenly became markedly breathless. The pulse was rapid and irregular and an electrocardiogram showed numerous multifocal ventricular ectopic beats. Potassium chloride and procaine amide were given, but death occurred a few hours later. All the murmurs persisted throughout the illness.

At necropsy (Dr. A. Schmaman) the heart weighed 705 G. with gross hypertrophy and dilatation of all 4 chambers. The myocardium was 7 mm. thick at the right ventricular outflow tract and 13 mm. thick at the outflow tract of the left ventricle. The endocardium appeared normal, except for a small area of thickening measuring 1 × 2 cm. immediately below the aortic valve. The aortic and pulmonary valves were normal. The mitral and tricuspid valves easily admitted 3 and 4 fingers respectively, and the valve curtains showed no macroscopic lesion. The coronary arteries, aorta, venae cavae, and iliac vessels were normal. The pulmonary arteries were healthy and there was no evidence of pulmonary infarction.

Histological examination of the ventricular myocardium showed hypertrophy of the myocardial fibres with some increase in the interstitial fibrous tissue. In a few areas there was slight infiltration of the interstitial tissue by polymorphonuclear leukocytes and lymphocytes. The left atrial appendage showed antemortem thrombus adherent to the endocardium. Aschoff nodes were not found. All the valve cusps and the aorta were histologically normal.

The pathological features were those of idiopathic cardiomyopathy.

Case 2

P.G., an African male aged 28 years, was admitted in January 1959 with weakness of the right side of the body and inability to speak. This was of acute onset and he had previously been quite well.

On examination the patient had a right hemiplegia and aphasia. The temperature was 101°F. and the pulse rate was 96 per minute. The pulse was distinctly collapsing in character, with a blood pressure of 135/40 mm.Hg. The heart was markedly enlarged with heaving impulses over both left and right ventricles. At the apex a pansystolic blowing murmur and a mid-diastolic rumble were audible. At the aortic area there was a long blowing diastolic murmur, commencing immediately after the second heart sound. The murmur was also audible down the left sternal border. A short mid-systolic ejection murmur was heard at the aortic area.

Radiography confirmed the generalized gross cardiomegaly. The aortic knuckle appeared normal. The lung fields were congested. Electrocardiography showed left ventricular predominance (S in V_2 + R in V_6 = 43 mm.). The haemoglobin level was 15·7 G. per 100 ml. and the leukocyte count was 9,200 per c.mm. The ESR was 56 mm. in the first hour (Wintrobe), but C-reactive protein was absent. The antistreptolysin O titre was 1,000 units per ml. Blood cultures yielded no growth after prolonged incubation. Urinalysis was normal.

The diagnosis of aortic and mitral incompetence of rheumatic origin was made and, in view of the neurological signs suggesting a cerebral embolus and the pyrexia, the patient was treated for subacute bacterial endocarditis.

Improvement was rapid and within 1 week the power of speech and movement had largely returned. Seven weeks later, however, the patient suddenly developed congestive cardiac failure. The antistreptolysin O titre was now 1,600 units per ml., and the C-reactive protein was positive, ++. Response to digitalis and diuretics was satisfactory. Three weeks later, however, the congestive cardiac failure recurred and, after partial improvement, the patient's condition deteriorated rapidly and he died. The murmurs persisted throughout the 3-month course of the illness.

At necropsy (Dr. I. Spector) the heart weighed 507 G. There was marked hypertrophy of the left and right ventricular walls, which measured 18 mm. and 7 mm. in thickness respectively. The ventricles were also dilated. The mitral valve admitted the tips of 3 fingers and the tricuspid valve 4 fingers. The valve cusps were normal. The aortic and pulmonary valves, aorta, pulmonary artery, and coronary arteries were likewise normal. Numerous mural thrombi were present in both ventricles and in the right atrial appendage. A thrombus was present in the right femoral vein. The lungs were mildly oedematous and showed several areas of recent infarction. The brain showed an area of softening in the left internal capsule.

Histological sections of the myocardium showed hypertrophy of the muscle fibres. The endocardium was normal and no Aschoff nodes were found.

The pathological features were those of idiopathic cardiomyopathy with mural thrombi, right femoral-vein thrombosis, and cerebral and pulmonary infarction.

DISCUSSION

The 2 cases described were found at necropsy to be examples of the idiopathic cardiomyopathy commonly seen in the Johannesburg African population.[2-4] The heart valves and aortas were normal. Yet, clinically, they presented as cases of organic valvular disease. Case 1 had the murmurs and thrills of both aortic stenosis and mitral incompetence, the murmur of aortic incompetence, and a poor pulse volume, while case 2 had the murmurs of aortic and mitral incompetence and a collapsing pulse. The murmurs persisted throughout the clinical course, and in case 2 were clearly audible both in the presence and absence of heart failure. The diagnosis of idiopathic cardiomyopathy, a condition with which we are thoroughly familiar at Baragwanath Hospital, was not

even remotely suspected. In view of the normality of the valves at autopsy, all the signs of valvular disease must have been functional in origin and the question of their mechanism arises.

Functional mitral incompetence with an apical pansystolic murmur is common in the cardiomyopathies, and is generally attributed to dilatation of the mitral ring. Mitral diastolic rumbles have also been described,[6] although in our experience they are rare. They may be caused by the association of left ventricular dilatation with a normal mitral ring, producing, as it were, a relative mitral stenosis.[7] Alternatively they may arise when the cardiomyopathy is associated with functional incompetence of the mitral or aortic valves. In case 2, in which a mid-diastolic rumble was heard, both valves were functionally incompetent. In organic mitral incompetence a mid-diastolic rumble is common, and is thought to be caused mainly by the increased blood flow through the mitral valve during the phase of rapid ventricular filling.[8] The factor of turbulence created by the diseased valve cusps *per se* is regarded as being of secondary importance. Functional mitral incompetence must also be associated with an increased atrio-ventricular flow in mid-diastole, and it is conceivable that on occasion this flow may be so large that it gives rise to a mid-diastolic rumble despite the absence of diseased mitral cusps. In organic aortic incompetence, mid-diastolic or presystolic rumbles of functional origin may occur,[9] the presystolic murmurs being classically associated with the name of Austin Flint.[10] Since these murmurs are thought to be caused by interference with mitral-valve function by regurgitating blood,[9] there is no reason why they should not also occur in cases of functional aortic incompetence.

The aortic stenotic murmur of case 1 may be explained by the observations of Brock.[11,12] He was the first to describe the occurrence of functional obstruction at the outflow tract of the left ventricle secondary to extensive myocardial hypertrophy. He pointed out that the walls of the lower part of the outflow tract of the left ventricle are chiefly muscular and that, when sufficiently hypertrophied, they may come together in early systole and obstruct the further emptying of the ventricle. Brock[12] reported 4 proved cases of this condition. All presented with the clinical features of aortic stenosis and had been referred to him for aortic valvotomy. Left ventricular pressure tracings, obtained either at operation or by percutaneous catheterization, demonstrated an obstruction at the level of the outflow tract. At operation or necropsy no evidence of organic stenosis, valvular or subvalvular, was found. In all, the principal finding was marked myocardial hypertrophy. In all but one, the aetiology of the myocardial hypertrophy was entirely obscure, the exception being a case in which systemic hypertension was thought to be the cause. Following Brock's description, there have been a number of further reports of left ventricular outflow obstruction secondary to idiopathic myocardial hypertrophy.[13-15] Our first case was characterized by massive biventricular hypertrophy of unknown cause, and it is therefore possible that the aortic stenotic murmur heard in this patient was also caused by functional obstruction of the left ventricular outflow tract. The associated murmur of aortic incompetence might possibly be explained on the basis of post-stenotic dilatation involving the aortic ring, as is known to occur with subvalvular organic stenosis.[16]

The free, functional aortic incompetence, which was present in case 2, has also been previously described in patients with cardiomyopathy.[17,18] Anders,[17] in particular, reported a number of cases and reviewed the literature critically. He attributed the incompetence to left ventricular dilatation. The mechanism he suggested was based on the anatomical studies of MacCallum[19] who demonstrated a sphincter-like band of muscle surrounding the left ventricular outflow tract. Anders postulated that in left ventricular dilatation this sphincter relaxed and so gave rise to dilatation of the outflow tract and the aortic ring, with resultant aortic incompetence. In support of this contention, Anders quoted experiments in dogs in which section of the sphincter resulted in immediate aortic incompetence. It would, however, be generally agreed that functional aortic incompetence is rarely found in left ventricular dilatation of cardiomyopathic or other origin; this may be related to the fact that the upper part of the left ventricular outflow tract (the aortic vestibule) and its continuation, the aortic ring, are relatively rigid structures consisting of tough fibrous tissue. MacCallum's sphincter surrounds only the lower portion of the outflow tract and this, as stated above, is chiefly muscular, and therefore much less rigid in structure. Dilatation following relaxation of the sphincter would therefore be expected to involve mainly the lower part of the tract, with considerably less effect on the upper part.

SUMMARY

Two unusual cases of idiopathic cardiomyopathy are described. They were characterized by persistent murmurs, diastolic as well as systolic, which suggested organic disease of the aortic and mitral valves. All the valves, however, were found to be normal at necropsy. The mechanism of production of these murmurs is discussed.

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Medical Association of South Africa

FORTY-THIRD SOUTH AFRICAN MEDICAL CONGRESS

24 – 30 SEPTEMBER 1961, CAPE TOWN

Telephone: 3-3355
Telegrams: 'MEDKONGRES'
Cape Town

THIRD AND FINAL CIRCULAR

Medical Congress Office
P.O. Box 643
35 Wale Street
CAPE TOWN

GENERAL INFORMATION AND FINAL PROGRAMME

The Forty-third Medical Congress of the Medical Association of South Africa will be held in *Cape Town* on *24 - 30 September 1961* at the University of Cape Town, Rondebosch, Cape

OFFICE BEARERS

PATRON

State President C. R. Swart

PRESIDENT OF CONGRESS

The President of the Medical Association of South Africa:
Mr. J. A. Currie

VICE-PRESIDENT OF CONGRESS

Dr. J. C. Coetzee

HONORARY VICE-PRESIDENTS

The Honourable, the Minister of Health, Dr. Albert Hertzog
The Honourable, the Administrator of the Cape Province, Mr. J. N. Malan
His Worship, the Mayor of Cape Town
The Chairman, Cape Divisional Council, Cllr. J. F. Blanckenberg
The President, South African Medical and Dental Council, Prof. S. F. Oosthuizen
The President, College of Physicians, Surgeons and Gynaecologists of South Africa, Dr. J. A. Douglas
The Past President, College of Physicians, Surgeons and Gynaecologists of South Africa, Prof. G. A. Elliott
The Chairman, Federal Council (M.A.S.A.), Dr. E. W. Turton
The Past Chairman, Federal Council (M.A.S.A.), Dr. J. H. Struthers
Dr. A. J. Orenstein, Life Vice-President of the Medical Association
Dr. J. H. Harvey Pirie, Life Vice-President of the Medical Association
Dr. A. W. S. Sichel, Life Vice-President of the Medical Association
The Principal, University of Cape Town, Mr. J. P. Duminy
The Rector, University of Stellenbosch, Prof. H. B. Thom
The President, South African Dental Association, Dr. J. F. v. d. S. de Villiers
The President, Pharmaceutical Society of South Africa, Mr. J. Sapire
The President, South African Nursing Council, Miss A. S. Latsky
Mrs. J. Newton Thompson
Prof. M. R. Drennan
Dr. T. Shadick Higgins
Dr. H. Kramer
Dr. J. Luckhoff
Dr. D. P. Marais
Dr. W. Lennox Gordon

ORGANIZING COMMITTEE

Executive Committee:

Chairman: Prof. B. Bromilow-Downing
Vice-chairman: Dr. J. C. Coetzee
Treasurer: Dr. J. G. Louw
Organizing Secretaries: Dr. Jacques Roux, Dr. G. S. Mulier Botha
and Drs. J. A. Currie, A. Landau, A. I. Goldberg, J. D. Joubert, A. H. Tonkin, A. P. Blignault, F. E. Hofmeyr and Mr. M. J. de Kock

Additional Members of Organizing Committee:

Mesdames V. Ferreira, J. D. Joubert, Profs. J. F. Brock, J. N. de Villiers, J. H. Louw, H. W. Weber, Drs. R. D. H. Baigrie, L. Blumberg, J. K. de Kock, B. J. v. R. Dreyer, R. Lane Forsyth, H. O. Hofmeyr, J. J. du Pré le Roux, J. R. E. Lee, N. Levy, D. McKenzie, F. K. Mitchell, F. W. F. Purcell, R. L. Retief, P. J. M. Retief, T. Schrire, J. J. W. van Zyl, J. J. van Heerden, A. A. Zabow

Sub-Committees:

Accommodation: Dr. J. C. Coetzee (convener)

Brochure and Publications: Dr. A. P. Blignault (convener), Drs. A. H. Tonkin, R. L. Kleinman, Mr. M. J. de Kock

Press and Publicity: Dr. A. P. Blignault (convener), Dr. R. L. Kleinman

Medical and Surgical Products Exhibits: Mr. M. J. de Kock

Entertainments: Dr. R. D. H. Baigrie (convener), Dr. H. W. Clegg (convener — Ball), Drs. S. T. de Kock, D. P. de Villiers, Mesdames A. B. W. Ferreira, J. D. Joubert, Drs. J. G. Louw, D. P. Marais, A. te W. Naude, R. Milner (convener — golf), E. B. Malherbe (convener — racing), J. H. Bam and W. Wilkie (conveners — tennis), P. C. W. Madden (convener — swimming), F. Visser and R. Lipschitz (conveners — fishing), D. Barnes (convener — squash), A. Katz (convener — bowls)

Ladies' Entertainments: Mrs. A. B. W. Ferreira (convener), Mrs. J. P. Roux (secretary), Mrs. J. A. Currie (treasurer), Mesdames J. F. Brock, A. J. Brink, J. N. de Villiers, H. O. Haarburger, H. O. Hofmeyr, F. E. Hofmeyr (convener — hairdressing), W. P. U. Jackson (convener — bridge), B. Kay (convenor — bowls), N. Levy, J. H. Louw, P. C. W. Madden (convener — tennis), P. J. M. Retief, M. Reznek, R. J. Smit (convener — golf), T. Schrire, I. Waynik, H. G. L. Rauch (convener — Ball floral decorations)

Transport: Mrs. J. D. Joubert (convener), Mesdames G. Dall, P. S. Jenkin, J. N. Abelsohn, D. Wells, A. Singer, M. Owen-Smith, P. Schulze, S. Leinberger, J. Smith, A. Bosman, J. D. C. Sim, V. Bell, J. H. Bam, T. de Kock, B. S. Jones, M. Dry, L. M. van der Spuy, D. Watson-Smith

Hobbies: Dr. T. Schrire (convener), Dr. F. K. Mitchell

Scientific Exhibits: Dr. H. O. Hofmeyr (convener), Drs. T. J. Dry and C. L. Wicht (joint secretaries), Drs. W. P. U. Jackson and H. Muller (members)

Students' Committee: Mr. R. Cowie (chairman), Messrs. E. Simson, G. Morrison, P. Brink and J. D. Godlonton

SCIENTIFIC COMMITTEE

Chairman: Dr. A. Landau
Secretary: Dr. G. S. Muller Botha

Office-bearers of Scientific Sections:

	Chairman	*Vice-chairman*	*Secretary*
Anaesthesia	Dr. O. V. S. Kok, Pretoria	Dr. H. V. Hankins, East London	Dr. P. A. Foster, Cape Town
Dermatology and Venereology	Dr. J. J. Jacobson, Cape Town	Dr. Jean Walker, Durban	Dr. S. Stein, Cape Town
General Practice	Dr. L. O. Vercueil, Maraisburg, Tvl.	Dr. A. G. Blyth, Ladismith, Cape	Dr. F. E. Hofmeyr, Cape Town
Hospital Administration	Dr. J. G. Steyn, East London	Dr. J. G. Burger, Cape Town	Dr. K. H. Field, Cape Town
Industrial and Military Medicine	Dr. W. Waks, Pretoria	Dr. B. Serebro, Johannesburg	Commdt. O. du Preez, Cape Town
Medicine	Dr. N. A. Rossiter, Durban	Dr. P. Leftwich, Johannesburg	Dr. A. Swanepoel, Cape Town
Obstetrics and Gynaecology	Dr. P. Connan, Bloemfontein	Dr. F. Daubenton, jnr., Johannesburg	Dr. P. F. M. du Toit, Bellville
Ophthalmology	Dr. N. L. Murray, Pretoria	Dr. E. T. Meyer, Johannesburg	Dr. S. Sacks, Cape Town
Orthopaedics	Mr. A. Lewer Allen, Durban	Mr. G. F. Dommisse, Pretoria	Mr. M. Singer, Cape Town
Otorhinolaryngology	Dr. L. E. Lane, Port Elizabeth	Dr. J. H. Casewell, Pretoria	Dr. D. V. Maytham, Cape Town
Paediatrics	Dr. F. P. Walt, Durban	Dr. E. Fasser, Pretoria	Dr. R. F. Maggs, Cape Town
Pathology	Dr. D. Ordman, Johannesburg	Prof. H. W. Weber, Bellville	Dr. C. E. Watson, Cape Town
Physical Medicine	Dr. C. Adler, Johannesburg	Dr. C. W. Coplans, Cape Town	Dr. H. T. van Diggelen, Cape Town
Psychiatry, Neurology and Neuro-surgery	Dr. K. Lewer Allen, Johannesburg	Dr. R. W. S. Cheetham, Durban	Dr. J. S. du T. de Wet, Cape Town
Public Health	Dr. A. Stephen, Durban	Dr. P. E. Richter, Port Elizabeth	Dr. P. Boekstein, Cape Town
Radiology	Dr. J. Muir Grieve, Cape Town	Dr. T. Fichardt, Pretoria	Dr. J. A. Beyers, Bellville
Surgery	Mr. W. Kark, Johannesburg	Mr. R. Lane Forsyth, Cape Town	Mr. J. E. Gasson, Cape Town
Thoracic Surgery	Mr. P. E. Marchand, Johannesburg	Mr. Denis Fuller, Johannesburg	Mr. G. S. Muller Botha, Cape Town
Urology	Mr. D. R. Barnes, Cape Town	Mr. C. D. Kisner, Johannesburg	Mr. P. J. M. Retief, Cape Town

GENERAL INFORMATION

The Congress Office will be located in the SRC Room, 1st Floor, Students' Union, University of Cape Town. Telephone: 69-8961 (10 lines).

Information Bureau: This will, as in the past, be conducted by Caltex (Africa) Limited, and will be housed in the *Varsity* Room at the south-east corner of the ground floor of the Students' Union.

Badges: Membership badges of distinctive colours will be issued as follows:

Congress officials	**Red**
Overseas visitors	**Purple**
Local members	**Royal Blue**
Local ladies	**Light Blue**
Visiting members	**Dark Green**
Visiting ladies	**Light Green**
Ladies Entertainment Committee	**Pink**
Medical exhibitors	**Orange**
Technicians	**Yellow**
Students	**Emerald Green**

Postal and Telegraphic Facilities: The Department of Posts and Telegraphs has kindly consented to establish a postal agency at Congress and this will be housed in the Upper Sports Hall. Full facilities for the delivery and clearance of mail, acceptance and delivery of telegrams, and sale of stamps will be provided. The post office will be open from 9 a.m. to 1 p.m. and 2 p.m. to 5 p.m.

Postal address: Medical Congress, University of Cape Town, Rondebosch.

Telegraphic address: MEDKONGRES, Cape Town. Telephone No. 69-8961 (10 lines).

Banking Facilities: Standard Bank, Barclays Bank, Volkskas and Netherlands Bank have each kindly consented to establish an Agency. Normal banking hours. These agencies will also be housed in the Upper Sports Hall.

Academic Dress: It is suggested that those who wish to do so may wear Academic Dress to the Church Service on Sunday evening, 24 September, and the Opening Ceremony on Monday evening. Members who require to hire Academic Dress are kindly requested to make their own arrangements with the Registrar, UCT, Lovers' Walk, Rondebosch.

Transport: Adequate transport for all official excursions will be provided. Details will be available at the Information Bureau. Arrangements will also be made to meet individual requirements.

Catering: Teas will be served, free of charge, at 10.45 - 11.15 a.m. and 3.30 - 4 p.m. in the Cafeteria and adjoining Staff Dining Room on the ground floor in the Students' Union.

Lunches will be served from 1 - 2 p.m. daily at a charge of 50c.

Rest Rooms: The following rest rooms are being made available on the First Floor in the Students' Union: Rhodes Room for men and Falconer Room for ladies.

ENTERTAINMENT AND SPORT

Clubs: Members of Congress are welcome at any time during Congress Week at the following clubs, which have extended facilities of honorary membership: Mowbray Golf Club, Westlake Golf Club, Royal Cape Golf Club, Rondebosch Golf Club, Metropolitan Golf Club, Milnerton Golf Club, King David Country Club, Keurboom Sports Club, Clovelly Country Club, Royal Cape Yacht Club, Zeekoevlei Yacht Club, Cape Aero Club, South African Turf Club (Kenilworth), Durbanville Turf Club, City Club, Alexandra Club (Ladies), Klub Here Sewentien, Civil Service Club.

Members are kindly requested to wear their official Congress Badges when presenting themselves at these clubs.

Sports Day: This will be Thursday, 28 September.

Men's Golf Competition: The afternoon of Thursday, 28 September has been set aside for the *Campbell-Watt Trophy* (convener — Dr. R. Milner; holder — Dr. Denis Hansen). The men's golf tournament will be played at the Royal Cape and Mowbray golf courses, in view of the fact that a very large entry of well over 100 is expected. Members are earnestly requested to signify at registration or at the Congress Office, SRC Room, before Thursday where they would like to play, i.e. at Mowbray or Royal Cape, and are asked to present themselves at the club of their choice between 1 and 2 p.m.

Ladies Golf Competition: (convener — Mrs. R. J. Smit). *Border Cup* (holder — Mrs. Beatrice Joubert). The Ladies' Golf Competition will be played at Mowbray Golf Course at 9 a.m. on Thursday, 28 September, and will take the form of an individual Stableford Competition. Ladies wishing to take part are earnestly requested to signify their intention at registration or at the Congress Office as soon as possible. Ladies who do not have their own transport are requested to signify accordingly when handing in their names.

Bowls: (conveners — Dr. A. Katz and Mrs. B. Kay). *Bristol Bowl* (holder — Dr. A. Z. Butt), *Casewell-Struthers Trophy* (holder — Dr. F. W. P. Cluver).

The Ladies' Bowls will be played at Milnerton Bowling Club at 9.30 a.m. on Thursday, 28 September. Ladies requiring transport are requested to assemble at the Jameson Hall, UCT, at 9 a.m.

The Men's Bowls competition will be played at Milnerton Bowling Club at 2.15 p.m. on Thursday, 28 September.

The playing of the Bowls Competitions is subject to weather permitting.

Racing: (convener — Dr. E. B. Malherbe). The following meetings are being held at Congress time: 23 September at 1.30 p.m. — Kenilworth; 27 September at 1.30 p.m. — Durbanville; and 30 September at 1.30 p.m. — Kenilworth. Honorary membership facilities have been extended to members of Congress for the purpose of attending these race meetings. Special Membership Badges will be issued at registration to those who wish to attend. Wearing of the Congress Membership Badge will *not* be enough to gain admission to the Members' Enclosures at these race meetings.

Fishing: (conveners — Drs. F. Visser and R. Lipschitz). Members who would like to sample rock angling or deep-sea fishing will be able to do so in small parties and under guidance of experts, provided the weather is favourable.

Tennis: (conveners — Dr. J. H. Bam and Mr. W. Wilkie). Courts are being made available at Kelvin Grove, King David Country Club, Keurboom Sports Club, and the University of Cape Town, as well as privately.

Swimming: (convener — Mr. P. C. W. Madden). Quite a few members have expressed the wish to take part in aquatic sports, and the University Baths will be made available.

Squash: (convener — Dr. D. Barnes). The University of Cape Town courts will be made available.

Individual conveners can be contacted through the Congress Office.

MEETINGS AND ENTERTAINMENTS
(Daily Programme)

The following are the official evening programmes during Congress Week:

Sunday Night: Church Service. The Diocesan College Chapel, Rondebosch, should prove to be an admirable setting for the interdenominational church service, which will commence at 6.45 p.m. Roads will be signposted and there is ample parking space in the college grounds. The choir will be in attendance. Academic dress may be worn — this is entirely optional.

Public Lecture. This will follow the church service and will be delivered at 8.30 p.m. by Prof. J. F. Brock in the New Science Lecture Theatre at the University of Cape Town, Rondebosch. The President of Congress, Mr. J. A. Currie, will preside.

Monday Night: The Opening Ceremony in the City Hall will be followed by a Civic Reception in the Drill Hall where members will be guests of the Mayor, Councillor A. H. Honikman, and the President of Congress, Mr. J. A. Currie, as joint hosts. This will take the form of a buffet supper.

Tuesday Night: The popular show Caltex 'Kaskenades' will be presented in the Zionist Hall, Hope Street, Cape Town, from 8.15 to 10 p.m. This is expected to be a very popular entertainment. Members are requested to signify their intention of attending the show as soon as possible. Tickets are obtainable at registration or at the Congress Office.

Wednesday Night: This is the night of the Ball in the City Hall. Dress Formal. The winner of the Triumph Herald competition on behalf of the Benevolent Fund of the Medical Association will be announced at the Ball and the car will be on the stage.

Thursday Night: The Congress Banquet at the Weizmann Hall is open to members *and their wives.*

As an alternative entertainment the Caltex Show is again being presented at the Zionist Hall, Hope Street, Cape Town, for those who do not wish to attend the Banquet and who were not able to see the show on Tuesday night. Members are requested to signify their intention of attending the show as soon as possible. Tickets are obtainable at registration or at the Congress Office.

Tickets for the Ball and the Banquet: Members are urged to buy their tickets upon registration, since it is evident from the response indicated in the Intention Forms that both these functions are going to be very well patronized and accommodation will be limited.

Price of Tickets: Ball R6.00 double. Banquet R9.00 double R5.00 single.

Friday Night: A Malay Choir and Khalifa Performance will be presented by courtesy of the Cape Malay Choir Board, commencing at 8 p.m., at the Woodstock Town Hall. Tickets (25 cents per person) are obtainable on registration or at the Congress Office. Accommodation at the Woodstock Town Hall is limited, and members are urged to obtain the tickets early, since the Khalifa has in the past always proved a popular attraction.

PROGRAMME OF ENTERTAINMENTS FOR LADY VISITORS

An attractive programme has been arranged and Ladies will be issued with a diary on registration and will be asked to signify which excursions and functions they wish to attend. The full programme is as follows:

Sunday, 24 September 1961:

Morning:	Registration, Students' Union — 10 a.m. - 5 p.m.
Afternoon: 6.45 p.m.	Interdenominational church service in the Diocesan College Chapel, Rondebosch.
Evening: 8.00 p.m.	Public Lecture by Prof. J. F. Brock in the New Science Lecture Theatre, University of Cape Town, Rondebosch.

Monday, 25 September 1961:

Morning:	Registration, Students' Union — tea will be served.
Afternoon: 2.00 p.m.	Assemble at Jameson Hall. Proceed to Enterprise Bakery, Claremont.

Conducted tour through factory and tea. Return at 5.00 p.m.

or

Proceed to Service Products (sheltered employment), N'dabeni. Conducted tour and tea.

Either tour may also include a visit to Yardley's Cosmetics Packaging Factory or Humphries' Sweet Factory.

[Convener: Mrs. O. M. Haarburger (Wynberg - Simonstown area).]

Tuesday, 26 September 1961:

Morning: Free.

Afternoon: 4.30 p.m. Mannequin Parade by Rejane's at the Clifton Hotel, with champagne cocktails to follow. Guests requiring transport may assemble at the Jameson Hall at 3.30 p.m.

[Convener: Mrs. Ivan Waynik (Cape Town - Camps Bay area).]

Wednesday, 27 September 1961:

Morning: Tour of the Lutheran Church and the Koopman's De Wet Museum; tea.

or

Visit to the Groote Kerk, followed by a tour of the Castle; tea.

(On both tours qualified guides will give commentaries.)

or

Trip up the cableway to Table Mountain summit; tea. A specially reduced fee of 8/- (80 cents) for the cableway is payable on arrival at the lower cableway station. This trip is subject to the weather being suitable.

For all these trips guests will assemble at the Jameson Hall at 9.30 a.m.

[Conveners: Mesdames J. H. Louw and P. C. W. Madden (Rondebosch - Kenilworth area).]

Afternoon: Free.

Thursday, 28 September 1961:

Morning: Golf, Tennis and Bowls competitions. Assemble at Jameson Hall at 9.00 a.m. Transport, where necessary, will be arranged to the respective sports grounds.

[Conveners: Mesdames Ryno Smit, P. C. W. Madden and B. Kay.]

Afternoon: 1.30 p.m. Assemble at Jameson Hall. Proceed to 'Vergelegen', the historic farm of Willem Adriaan van der Stel at Somerset West. Picnic tea with hostess drivers.

or

Assemble as above; proceed to 'Protea Heights', Stellenbosch. Tea at Stellenvale Wineries, Devon Valley.

or

Visit to KWV cellars at Paarl; tea as guests of the KWV.

[Conveners: Mrs. P. J. M. Retief (Pinelands-Milnerton area), and Mesdames A. Brink, J. de Villiers and M. Reznek (Bellville - Parow area).]

Friday, 29 September 1961:

Morning: 9.30 a.m. Assemble at Jameson Hall and proceed to Kirstenbosch for a tour of the gardens; tea.

or

Proceed to Groot Constantia, driving through the Constantia Valley; tea.

[Conveners: As on Thursday.]

Afternoon: Free.

OFFICIAL CONGRESS PROGRAMME

21 - 23 SEPTEMBER 1961

Meeting of Federal Council.

FRIDAY 22 SEPTEMBER 1961

9.30 a.m. Annual General Meeting of the Medical Association of South Africa.

SATURDAY, 23 SEPTEMBER 1961

1.30 p.m. Race meeting, South African Turf Club, Kenilworth (see general information).

SUNDAY, 24 SEPTEMBER 1961

9.00 a.m. Annual General Meeting, South African Society of Anaesthetists, Cecil Hotel, Main Road, Newlands.

12.45 p.m. South African Society of Anaesthetists, Annual Luncheon, same venue.

10 a.m. – 5 p.m. Registration in the Students' Union at the University of Cape Town.

6.45 p.m. Interdenominational Church Service, Diocesan College Chapel, Rondebosch.

6.30 p.m. Jewish Synagogue Service, Great Synagogue, Gardens.

8.30 p.m. Public Lecture (New Science Lecture Theatre, University of Cape Town) by Prof. J. F. Brock — 'Man and Medicine'.

MONDAY, 25 SEPTEMBER 1961

8.30 a.m. onwards Registration, Students' Union, University of Cape Town.

9.30 a.m. Opening of Trades Exhibition, Jameson Hall, by the President of Congress, Mr. J. A. Currie.

10.15 a.m. Opening of Hobbies Exhibition, Senate Room, Arts Block, by Prof. D. P. Inskip.

11.00 a.m. Opening of Scientific Exhibition, Zoology Department, by the President of the Cape Western Branch, Dr. A. Goldberg.

MONDAY AFTERNOON

Television (Monday):

New Science Lecture Theatre.

12.30 – 2.00 p.m. 'Repair of ventricular septal defect' — Dr. C. N. Barnard.

National Group Business Meetings (Monday):

2.15 p.m. District Surgeons — Arts Block, Room A.
Neuro-psychiatry — Arts Block, Room B.
Medical Officers of Health — Arts Block, Room C.
Surgery — Arts Block, Room D.

4.00 p.m. Thoracic Surgery — Arts Block, Room A.
Urology — Arts Block, Room C.
Otorhinolaryngology — Arts Block, Room E.
General Practice — Arts Block, Room F.

Ophthalmology (Monday):

Department of Architecture, Room A.

Chairman: Dr. N. Murray (Pretoria).

2.15 – 2.45 p.m. Prof. D. P. Choyce (London): Acrylic anterior chamber implants — Part I.

3.00 – 3.30 p.m. Prof. D. P. Choyce (London): Acrylic anterior chamber implants — Part II.

3.30 – 4.00 p.m. Tea interval.

4.15 – 4.35 p.m. Dr. E. Epstein (Johannesburg): Transciliary extraction of (1) persistent primary vitreous, and (2) posteriorly dislocated lenses.

4.45 – 5.00 p.m. Dr. J. L. van Selm (Cape Town): Results of lens extraction with general anaesthesia.

5.05 – 5.15 p.m. Dr. J. G. Louw (Cape Town): Demonstration of cases with anterior-chamber implants.

Otorhinolaryngology (Monday):

Department of Architecture, Room B.

Chairman: Dr. L. E. Lane (Port Elizabeth).

2.15 – 2.33 p.m. Dr. J. F. Jarvis (Cape Town): Climatic factors in vasomotor rhinitis.

2.41 – 2.59 p.m. Dr. G. H. Caiger (Durban): Inadequate fronto-nasal duct and migraine-type headache with a method of surgical treatment.

3.07 – 3.25 p.m. Dr. P. Meyrick (Pietermaritzburg): Tracheostomy.

Industrial and Military Medicine (Monday):

Department of Architecture, Room D.

Chairman: Dr. W. Waks (Pretoria).

2.15 – 2.40 p.m. Dr. L. F. Freed (Johannesburg): The principles of industrial psychology: An integralist approach.

2.40 – 3.15 p.m. Dr. R. C. Dickson (Dunnottar, Tvl.): A pilot survey of otitic conditions in Bantu mine labourers of the East Rand.

3.30 – 4.00 p.m. Tea interval.

4.00 – 4.20 p.m. Dr. D. I. Guthrie (Dynamite Factory, Somerset West): A rehabilitation unit.

4.20 – 4.40 p.m. Dr. I. Webster (Johannesburg): On a pneumoconiosis theme.

Urology, Radiology, Pathology (Monday):

Department of Architecture, Room C.

Chairman: Mr. D. R. Barnes (Cape Town).

2.15 – 2.30 p.m. Dr. W. G. Davis (Cape Town): Carcinoma of the prostate — pathological considerations.

2.30 – 2.45 p.m. Dr. T. Fichardt (Pretoria): Radiodiagnosis and radiotherapy of prostatic cancer.

2.45 – 3.00 p.m. Mr. J. C. Jordaan (Johannesburg): Treatment of carcinoma of the prostate.

Medicine (Monday):

Arts Block, Large Lecture Theatre.

Chairman: Dr. N. A. Rossiter (Durban).

2.15 – 2.30 p.m. Dr. R. S. Mibashan (Cape Town): The non-gouty relationships of serum uric acid — its significance in thrombosis, renal and blood disorders.

2.30 – 3.00 p.m. Dr. R. A. MacDonald (Boston, USA): Haemochromatosis and haemosiderosis in Boston.

3.00 – 3.15 p.m. Prof. L. Eales (Cape Town): Porphyria — the acute attack.

3.30 – 4.00 p.m. Tea interval.

Chairman: Dr. P. Leftwich (Johannesburg).

4.00 – 4.15 p.m. Dr. L. H. Klugman (Johannesburg): The value of the red-cell uptake of I^{131}-labelled triiodothyronine in the diagnosis of hyperthyroidism.

4.15 – 4.45 p.m. Prof. M. Rachmilewitz (Jerusalem): Nutritional anaemia.

4.45 – 5.00 p.m. Dr. B. G. Shapiro (Cape Town): Treatment of adipose gynandrism.

Obstetrics and Gynaecology (Monday):

Department of Land Surveying, Room A.

Chairman: Dr. P. Connan (Bloemfontein).

2.15 – 2.35 p.m. Dr. C. Craig (Cape Town): The obstetric flying squad and 'avertin' in the management of eclampsia.

2.40 – 3.00 p.m. Dr. N. Louw (Cape Town): The value of hypotensive drugs in the treatment of hypertension in pregnancy.

3.05 – 3.25 p.m. Dr. P. F. M. du Toit (Stellenbosch): Diabetes mellitus gedurende swangerskap.

3.30 – 4.00 p.m. Tea interval.

4.00 – 4.20 p.m. Dr. H. V. F. Jordaan (Cape Town): Comparative cranial morphology.

4.25 – 4.45 p.m. Dr. T. Fichardt (Pretoria): The Pretoria method in radiotherapy of cancer of the cervix uteri.

Anaesthesia (Monday):

Sectional Meeting for Moore-Dyke Registrar's Prize.

Department of Architecture, Room E.

Chairman: Dr. F. W. Roberts (Johannesburg).

Judges: Dr. G. Jackson Rees, Dr. R. P. Harbord, and Prof. O. V. S. Kok.

2.00 – 2.30 p.m. Dr. J. A. Cowlin (Stellenbosch): Halothane tachypnoea.

2.45 – 3.15 p.m. Dr. A. M. Whitaker (Cape Town): Chloroform anaesthesia and hypotension.

3.30 – 4.00 p.m. Tea interval.

4.00 – 4.30 p.m. Dr. J. Haumann (Stellenbosch): Antiemetic properties of halothane.

4.45 – 5.15 p.m. Dr. M. J. Louw (Stellenbosch): Mechanisms of liver toxicity with chloroform.

Orthopaedics (Monday):

Botany Building, Large Lecture Theatre.

Chairman: Mr. A. Lewer Allen (Durban).

2.15 – 2.30 p.m. Mr. Alec Singer (Cape Town): The treatment of coccydinia by sacro-coccygeal arthroplasty.

2.35 – 2.50 p.m. Mr. W. J. J. Thomas (Johannesburg): A new method of bone grafting ununited fractures of the carpal navicular — preliminary report.

2.55 – 3.10 p.m. Mr. Martin Singer (Cape Town): Quot homines, tot sententiae.

3.30 – 4.00 p.m. Tea interval.

4.00 – 4.20 p.m. Mr. D. S. C. Proctor (Cape Town): The functional treatment of fractures of the femur.

Hospital Administration (Monday):

Department of Land Surveying, Room B, Architecture Building.

Chairman: Dr. J. G. Steyn (East London).

2.15 p.m. Dr. J. H. Cairns (Cape Town): Modern trends in the planning of teaching hospitals.

4.00 p.m. Dr. E. R. Rudman (Cape Town): The rôle and organization of blood transfusion services in relation to the medical services in South Africa.

Paediatrics, Medicine, Allergy, General Practice (Monday):

Zoology Block, Large Lecture Theatre.

Chairman: Dr. A. G. Blyth (Ladismith, Cape).

2.15 – 2.30 p.m. Dr. Noel Storr (Cape Town): Asthma in South Africa — a research problem for the general practitioner.

2.30 – 2.55 p.m. Dr. D. Ordman (Johannesburg): Problems in the practice of allergy.

2.55 – 3.10 p.m. Dr. S. C. Shore (Cape Town): Treatment of childhood asthma by oral desensitization.

3.10 – 3.25 p.m. Dr. B. G. Shapiro (Cape Town): Control of chronic diarrhoea by restriction of bread in the diet.

Surgery, Thoracic Surgery, Medicine (Monday):

Psychology Building, Large Lecture Room.

Chairman: Mr. D. Fuller (Johannesburg).

2.15 – 2.30 p.m. Mr. W. Silber (Cape Town): Oesophageal hiatal hernia — the problem of diagnosis and treatment.

2.30 – 2.45 p.m. Mr. P. E. Marchand (Johannesburg): The relationship between trauma and hiatus hernia: a medico-legal and WCA problem.

2.45 – 3.00 p.m. Mr. G. S. Muller Botha (Cape Town): The advantages of high-volume suction and posture in drainage of the chest.

3.00 – 3.15 p.m. Mr. E. J. Joubert (Johannesburg): Infection in relation to the use of prostheses in cardiovascular surgery.

Neuro-psychiatry (Monday):
Chemistry Building, New Lecture Theatre.
Chairman: Dr. K. Lewer Allen (Johannesburg).

4.00 – 4.20 p.m. Dr. R. W. S. Cheetham (Durban): Adolescence today.
4.30 – 4.50 p.m. Dr. J. MacGregor (Cape Town): The meaning of pallidotomy.

Television (Monday):
New Science Lecture Theatre.

4.00 – 5.30 p.m. Paediatric programme — Dr. J. D. L. Hansen.

MONDAY EVENING

Opening Ceremony and Mayoral and Presidential Reception (City Hall, Cape Town).

8 p.m. Congress will be officially opened by Mr. J. P. Duminy, Principal of the University of Cape Town.

TUESDAY, 26 SEPTEMBER 1961
TUESDAY MORNING

Plenary Session: 'Care of the Aged' (Tuesday):
New Science Lecture Theatre.
Chairman: President of Congress, Mr. J. A. Currie (Cape Town).

9.10 – 9.25 a.m. Opener — Mrs. Zerilda Steyn (Chairman, South African National Council for the Welfare of the Aged, Cape Town): Allesomvattende versorging van bejaardes.
9.25 – 10.10 a.m. Dr. J. H. Sheldon (Great Britain): Old people and their health.
10.10 – 10.25 a.m. Dr. I. M. Hurwitz (Cape Town): The elderly and some of their disabilities.
10.45 – 11.15 a.m. Tea interval.
11.15 – 11.35 a.m. Adv. B. J. Vorster (Minister van Justisie): Die plek van Volkswelsyn in die versorging van bejaardes.
11.35 – 11.50 a.m. Dr. R. L. Retief (Director of Hospital Services — Cape Town): Die plek van die Provinsiale Administrasie in die versorging van bejaardes.
11.50 a.m. – 12.10 p.m. Mr. S. P. Watson (Secretary of the Rand Aid Association, Johannesburg): Rehabilitation of the aged.
12.10 – 12.20 p.m. Dr. R. Getz (Johannesburg): The growth of work at a Jewish aged home.

TUESDAY AFTERNOON

Group Business Meetings (Tuesday):

4.00 p.m. Pathology — Arts Block, Room B.
5.15 p.m. Endocrinology — Chemistry Lecture Theatre.
5.15 p.m. Physicians — Arts Block, Room D.

Combined Meeting: 'Care of the Aged' (Tuesday):
Psychology Building, Large Lecture Room.
Chairman: Dr. L. Blumberg (Cape Town).

2.15 – 2.30 p.m. Dr. S. Galloon (Cardiff): Anaesthesia in old people.
2.30 – 2.45 p.m. Dr. H. Bell (Stellenbosch): Fractures in the aged.
2.45 – 3.00 p.m. Mr. S. Scher (Cape Town): Urinary incontinence in the aged.
3.00 – 3.15 p.m. Mr. W. Silber (Cape Town): Aspects of geriatric surgery.
3.30 – 4.00 p.m. Tea interval.
4.00 – 4.20 p.m. Dr. R. W. S. Cheetham (Durban): Psychiatric aspects of care of the aged.
4.20 – 4.40 p.m. Dr. D. Glajchen (Johannesburg): The medical and social evaluation of the in-patient aged 65 years and over in the Johannesburg General Hospitals—a cross-sectional survey.
4.40 – 5.00 p.m. Dr. A. L. Lomey (Johannesburg): The home for aged and infirm Hebrews of New York.

Medicine, Gynaecology, Surgery, Urology, Paediatrics (Tuesday):
Symposium on 'Intersex':
Chemistry Building, New Lecture Theatre.

2.15 – 2.35 p.m. Dr. R. Hoffenberg (Cape Town): Chromosomes and sex.
2.35 – 2.55 p.m. Dr. B. Pimstone (Cape Town): Females with testes.
2.55 – 3.15 p.m. Mr. H. Katz (Cape Town) — Films: Surgical treatment of a true hermaphrodite.

Medicine, Urology, Surgery (Tuesday):
Symposium on 'Hypercalciuria':
Chemistry Building, New Lecture Theatre.

4.00 – 4.20 p.m. Dr. W. P. U. Jackson (Cape Town): Urine calcium as a regulator of calcium homeostasis.
4.20 – 4.40 p.m. Dr. E. B. Dowdle (Cape Town): The hypercalciuria of sarcoidosis.
4.40 – 5.00 p.m. Mr. M. Modlin (Cape Town): Renal stones and hypercalciuria.

Radiology (Tuesday):
Department of Architecture, Room B.
Chairman: Prof. J. M. Grieve (Cape Town).

2.15 – 2.30 p.m. Dr. T. Fichardt (Pretoria): Experience in the treatment of inoperable cancer with a 2,000 curie telecobalt unit.
2.30 – 3.15 p.m. Prof. H. Druckrey (Germany): Pharmacology of cytotoxic drugs.
3.30 p.m. Tea interval.
4.00 – 4.15 p.m. Dr. F. N. Gillwald (Welkom): Protection against radiation in the diagnostic X-ray department.
4.15 – 4.30 p.m. Dr. G. R. H. Sealy (Cape Town): Modern trends in radiotherapy.
4.30 – 4.45 p.m. Dr. J. N. Jacobson (Johannesburg): An additional cause for elevation of a diaphragm dome.

Paediatrics, Pathology (Tuesday):
Department of Land Surveying, Room A, Architecture Building.
Chairman: Dr. E. Fasser (Pretoria).

2.15 – 2.30 p.m. Dr. J. D. L. Hansen (Cape Town): The determination and significance of intestinal malabsorption in infancy and childhood.
2.30 – 2.45 p.m. Dr. H. Stein (Johannesburg): Veno-occlusive disease of the liver in Johannesburg.
2.45 – 3.00 p.m. Drs. G. M. Potgieter, P. M. Smythe, Prof. J. Kench (Cape Town): The serum proteins in kwashiorkor.
3.00 – 3.15 p.m. Drs. P. M. Smythe, A. Swanepoel, J. A. H. Campbell (Cape Town): The heart in kwashiorkor.

Paediatrics and Thoracic Surgery (Tuesday):
Department of Land Surveying, Room A, Architecture Building.
Chairman: Dr. F. P. Walt (Durban).

4.00 – 4.15 p.m. Prof. F. J. Ford: The clientéle of a children's hospital in Cape Town: a year's survey of the patients seeking advice, their distribution with regard to age, sex, and race, and the broad picture of their nutrition and the most common kinds of illness.
4.15 – 4.30 p.m. Dr. W. Emdin (Cape Town): Basic infant feeding.
4.30 – 4.45 p.m. Mr. G. S. Muller Botha (Cape Town): The management of partial thoracic stomach in infancy and childhood — a new concept.

4.45 – 5.00 p.m. Dr. L. S. Taitz (Johannesburg): Parathyroid function in vitamin-D deficiency rickets.

Anaesthesia (Tuesday):
Zoology Building, Large Lecture Theatre.
Chairman: Dr. H. V. Hankins (East London).

4.00 – 4.30 p.m. Dr. G. Jackson Rees (Liverpool): Anaesthesia for cardioangiography.
4.45 – 5.00 p.m. Dr. S. Galloon (Cardiff): Closed-circuit halothane.

Orthopaedics, Physical Medicine (Tuesday):
CAFDA Industrial Training Centre, Prince George Drive, Retreat.

2.00 – 4.00 p.m. Introductory talk: The functions and working of the Industrial Training Centre. Demonstration.

Hospital Administration (Tuesday):
Department of Architecture, Room C.
Chairman: Dr. J. G. Steyn (East London).

2.15 p.m. Dr. J. G. Burger (Cape Town): Hospitalization, housing and accommodation of the aged.
Dr. R. L. Kotze (Stellenbosch): Die operasiesaaltegnikus.

Chairman: Dr. J. G. Burger (Cape Town).

4.00 p.m. Dr. P. N. Swanepoel (Pretoria): A hospital administrator and his relationship to the advent of nuclear medicine.
Dr. T. Schrire (Cape Town): Organization for mass casualties.

Public Health and Dermatology (Tuesday):
Botany Lecture Theatre.
Chairman: Dr. A. Stephen (Durban).

2.15 – 2.45 p.m. Dr. A. J. Wilsen (Worcester): TB control in the rural areas of the Western Cape.
2.45 – 3.05 p.m. Dr. S. Stein (Cape Town): Streptomycin dermatitis in nurses.
3.30 – 4.00 p.m. Tea interval.
4.00 – 4.30 p.m. Dr. F. A. Donnolly (Lusaka, N. Rhod.): Introduction to socio-economic medicine.

Ophthalmology, Neuro-surgery (Tuesday):
Department of Architecture, Room E.
Chairman: Dr. N. L. Murray (Pretoria).

2.15 – 3.00 p.m. Prof. Algernon B. Reese (New York): Diagnosis and treatment of orbital tumours.
3.15 – 3.30 p.m. Dr. Eric Meyer (Johannesburg): Orbital tumours.
3.30 – 4.00 p.m. Tea interval
4.00 – 4.15 p.m. Drs. R. L. H. Townsend and J. G. Louw (Cape Town): Ocular and orbital neoplasms.
4.30 – 4.50 p.m. Dr. J. L. van Selm (Cape Town): Vertical ocular deviations treated by surgery.
4.55 – 5.10 p.m. Dr. S. Abel (Cape Town): Contact lenses.

Thoracic Surgery, Surgery, Cardiology (Tuesday):
Department of Architecture, Room D.
Chairman: Mr. P. E. Marchand (Johannesburg).

4.00 – 4.15 p.m. Dr. C. N. Barnard, Mr. M. Klein (medical student) (Cape Town): A hole in the heart.
4.15 – 4.30 p.m. Mr. D. Fuller, Dr. J. L. Braudo, Dr. E. Goldblatt and Dr. M. M. Zion (Johannesburg): Surgical experiences in congenital heart disease.
4.30 – 4.45 p.m. Dr. C. N. Barnard (Cape Town): The surgical approach to severe tetralogy of Fallot.
4.45 – 5.10 p.m. Dr. J. L. Braudo, Dr. M. M. Zion (Johannesburg): Ventricular septal defect.

Otorhinolaryngology (Tuesday):
Department of Architecture, Room A.
Chairman: Dr. L. E. Lane (Port Elizabeth).

2.15 p.m. Dr. Michel Portman (France): Deafness surgery.
Film.
3.30 – 4.00 p.m. Tea interval

Neuro-psychiatry (Tuesday):

5.00 p.m. Psychiatric case conference at Groote Schuur Hospital (Ward F2), Department of Neuro-psychiatry.

Television (Tuesday):
New Science Lecture Theatre.

4.00 – 5.30 p.m. 'Radical mastectomy' — Prof. J. H. Louw.

RMO Group Reception (Tuesday):

6.00 p.m. Cocktails and Buffet Supper at the home of Dr. W. P. Steenkamp, Tamboerskloof.

TUESDAY EVENING

8.15 p.m. Caltex 'Kaskenades', Zionist Hall, Hope Street, Cape Town.

WEDNESDAY, 27 SEPTEMBER 1961

WEDNESDAY MORNING

Plenary Session: 'Diabetes' (Wednesday):
New Science Lecture Theatre.
Chairman: President of Congress, Mr. J. A. Currie.
(To solicit questions for panel discussion on diabetes in afternoon.)

A. DIABETES IN AFRICA

9.10 – 9.20 a.m. Opener — Dr. W. P. U. Jackson (Cape Town).
9.20 – 9.35 a.m. Dr. G. D. Campbell and Dr. J. McKechnie (Durban): Recent observations on diabetes in Zulus and Natal Indians.
9.35 – 9.50 a.m. Dr. H. C. Seftel (Johannesburg): Diabetes in the African as seen in Johannesburg.
9.50 – 10.05 a.m. Mr. W. M. Politzer and Dr. T. Schneider (Johannesburg): Diabetes in rural and urban Africans.
10.05 – 10.20 a.m. Prof. J. A. Tulloch (Kampala): Diabetes — a comparison with other parts of the tropics.
10.45 – 11.15 a.m. Tea interval

B. ORAL AGENTS IN DIABETES

11.15 – 11.35 a.m. Dr. D. Jackson (Britain): Chemistry, pharmacology and indications for oral antidiabetic substances.
11.35 – 11.50 a.m. Dr. T. Schneider (Johannesburg): Clinical experiences with oral hypoglycaemic drugs.
11.50 a.m. – 12.05 p.m. Dr. P. E. S. Loubser (Karl Bremer): Clinical experiences with the oral hypoglycaemic drugs.
12.05 – 12.20 p.m. Dr. J. B. Herman (Cape Town): Glucose tolerance during treatment with hypoglycaemic agents.
12.20 – 12.45 p.m. Discussion — opener Dr. C. L. Wicht (Karl Bremer).

WEDNESDAY AFTERNOON

Group Business Meetings (Wednesday):

1.45 p.m. Christian Medical Fellowship — Arts Block, Room B.
2.15 p.m. RMO Group — Arts Block, Room B.

Combined Meeting: 'Diabetes' (Wednesday):
Psychology Building, Large Lecture Room.
Chairman: Prof. G. C. Linder (Cape Town).

2.15 – 2.30 p.m. Dr. C. L. Wicht: Peripheral vascular changes in diabetes.

2.30 – 2.50 p.m. Drs. I. N. Marks, S. Bank, W. P. U. Jackson: Pancreatic diabetes.
2.50 – 3.00 p.m. Dr. H. van der Meulen: Necrobiosis lipoidica diabeticorum.
3.00 – 3.15 p.m. Dr. D. Jackson: Wider implications of the sulphonyl ureas.
3.30 – 4.00 p.m. Tea interval

4.00 p.m. *Panel Discussion on Diabetes:*
Chairman and Moderator: Prof. G. C. Linder (Cape Town).
Members of Panel: Dr. W. P. U. Jackson, Dr. C. L. Wicht, Prof. J. H. Louw, Dr. I. Grayce, Prof. J. T. Louw, Miss. J. Dreosti, Dr. T. Schneider.

Hospital Administration and General Practice (Wednesday):
Botany Lecture Theatre.
Chairman: Dr. J. G. Burger (Cape Town).

2.15 p.m. Dr. J. D. Prestwich (Johannesburg): The utilization of general practitioner services in a Provincial Hospital.
Dr. J. A. Louw (Bethlehem, OFS): Administratiewe moeilikhede van die kleiner plattelandse Provinsiale hospitale.

Chairman: Dr. J. G. Steyn (East London).
4.00 p.m. Dr. H. R. Ackermann (Cape Town): Staphylococcal hospital cross-infection.

Neuro-psychiatry (Wednesday):
Department of Architecture, Room E.
Chairman: Dr. K. Lewer Allen (Johannesburg).

2.15 – 2.35 p.m. Dr. B. W. Levinson (Johannesburg): The hypnotist and the patient — who gets the treatment?
2.40 – 3.00 p.m. Dr. P. C. Keet (Cape Town): Traumatic intracerebral haematoma.
3.05 – 3.25 p.m. Dr. J. MacGregor (Cape Town): Atypical facial palsies.
Tea interval
4.00 – 4.20 p.m. Dr. B. J. F. Laubscher (Port Elizabeth): The nature of psychokinesis — a theory.
4.30 – 4.50 p.m. Dr. M. V. Bührmann (Cape Town): Contribution of psychiatry to maternal and child-welfare services.

Physical Medicine and Orthopaedics (Wednesday):
Department of Land Surveying, Room B, Architecture Building.
Chairman: Dr. C. Adler (Johannesburg).

2.15 – 3.15 p.m. Dr. A. L. Lomey (Johannesburg): Report on a visit to:
(*a*) 3rd International Congress of Physical Medicine, Washington;
(*b*) 8th World Congress of International Society for Welfare of Cripples, New York;
(*c*) Mayo Clinic.
Tea interval
4.30 p.m. Dr. C. W. Coplans (Cape Town): The use of sclerosant solutions in the treatment of low back pain.

Industrial and Military Medicine (Wednesday):
Department of Architecture, Room B.
Chairman: Dr. B. Serebro (Johannesburg).

2.15 – 2.40 p.m. Dr. L. F. Freed (Johannesburg): The problem of fatigue in industry — an integralist approach.
2.40 – 3.05 p.m. Dr. A. M. Coetzee (Johannesburg): Preventive industrial medicine on the gold mines.
Tea interval
4.00 – 4.20 p.m. Drs. M. E. Barry, B. King, J. G. D. Laing, J. G. Foster (Welkom, OFS): Classification of heat illness and description of the clinical features of heat exhaustion.
4.20 – 4.40 p.m. Drs. M. E. Barry, B. King, J. G. D. Laing, J. G. Foster (Welkom, OFS): The aetiology and clinical features of heatstroke.

Anaesthesia, Surgery, Medicine, Thoracic Surgery, Radiology, Urology (Wednesday):
Department of Architecture, Room D.
Chairman: Mr. W. Kark (Johannesburg).

2.15 – 2.30 p.m. Drs. J. Terblanche, L. C. Isaacson, Prof. L. Eales and Dr. C. N. Barnard (Cape Town): Renal function during and immediately after profound hypothermia.
2.30 – 2.45 p.m. Dr. J. K. Bremer, Dr. J. E. Combrinck (Pretoria): Diep hipotermie deur middel van gedeeltelike ekstra-korporale sirkulasie met hitte-uitruiling by skape.
2.45 – 3.00 p.m. Dr. B. Goldberg, Dr. N. W. Levin, Mr. H. Gaylis, Mr. E. Lipworth, Dr. R. Glyn-Thomas (Johannesburg): Hypertension and unilateral kidney disease.
3.00 – 3.15 p.m. Dr. B. Kottler (Cape Town): Experiences with renal arteriography.
3.30 – 4.00 p.m. Tea interval.

Urology, Radiology (Wednesday):
Department of Architecture, Room D.
Chairman: Mr. C. D. Kisner (Johannesburg).
4.00 p.m. Dr. C. Komins and Mr. J. Jordaan (Johannesburg): Bladder-neck obstructions — a demonstration of the rôle afforded by cine-urethrocystography in the treatment.

Ophthalmology, Paediatrics, Pathology (Wednesday):
Department of Architecture, Room A.
Chairman: Mr. E. T. Meyer (Johannesburg).

2.15 – 2.45 p.m. Prof. Algernon B. Reese (New York): Paediatric ophthalmology.
3.00 – 3.15 p.m. Drs. J. Rabkin and S. C. Shore (Cape Town): Toxoplasmosis — clinical manifestations in children.
3.15 – 3.20 p.m. Dr. J. H. S. Gear (Johannesburg): Serological tests for toxoplasmosis.
Tea interval.

Ophthalmology (Wednesday):
Department of Architecture, Room A.
Chairman: Mr. E. T. Meyer.
Symposium on Photocoagulation.

4.00 – 4.30 p.m. Dr. G. L. Trichard (Bloemfontein): Photocoagulation.
4.30 – 4.50 p.m. Dr. Wallace M. Levy (Johannesburg): Photocoagulation.
5.05 – 5.15 p.m. Dr. S. Abel (Cape Town): Experiences with EDTA.

Public Health (Wednesday):
Department of Architecture, Room C.
Chairman: Dr. A. Stephen (Durban).

2.15 – 2.40 p.m. Dr. R. M. Langerman (Cape Town): Typhoid fever in a large urban area.
2.40 – 3.10 p.m. Dr. J. G. D. Laing (Welkom): An outbreak of smallpox in a semi-closed community.
Tea interval.
4.00 – 4.30 p.m. Dr. H. Bloomberg (Brakpan): A rheumatic fever clinic — a contribution to a public health problem.

Orthopaedics (Wednesday):
Lady Michaelis Orthopaedic Hospital, Plumstead.
Chairman: Mr. H. Bell (Stellenbosch).
2.00 – 4.00 p.m. Clinical demonstration.

Anaesthesia (Wednesday):

Arts Block, Large Lecture Theatre.

Chairman: Dr. J. Abelsohn (Cape Town).

4.00 – 4.20 p.m. Dr. S. V. Potgieter (Bloemfontein): Anaesthesia in scoliosis.

4.30 – 4.50 p.m. Dr. W. van der M. Lambrechts (Cape Town): The anaesthetist and postoperative pain.

Otorhinolaryngology (Wednesday):

Department of Land Surveying, Architecture Building, Room A.

Chairman: Dr. R. L. E. Lane (Port Elizabeth).

2.15 – 2.35 p.m. Dr. B. T. Bernstein (Johannesburg): Injury to the auditory ossicular chain and its repair.

2.45 – 3.00 p.m. Dr. I. A. van N. Fick (Pretoria): Dekompressie van die labirint — chirurgiese ingreep vir Meniere se siekte.

3.07 – 3.25 p.m. Dr. A. Schiller (Cape Town): Mastoid osteoplasty.
Tea interval.

4.00 – 4.20 p.m. Dr. C. J. du Toit (Cape Town): The graft in reconstructive middle-ear surgery.

Paediatrics, Medicine, Pathology (Wednesday):

Chemistry Building, New Lecture Theatre.

Chairman: Dr. G. Selzer (Cape Town).

4.00 – 4.15 p.m. Drs. H. Malherbe, E. Kahn, R. Cassel and P. Roux (Johannesburg): The rôle of enteropathogenic strains of *E. coli* and viruses in diarrhoeal disorders in Johannesburg.

4.15 – 4.30 p.m. Dr. H. de V. Heese (Cape Town): Staphylococcal empyema in infancy and childhood.

4.30 – 5.00 p.m. Dr. J. H. S. Gear (Johannesburg): Virus diseases of the newborn.

Surgery, Thoracic Surgery, Medicine (Wednesday):

Zoology Lecture Theatre.

Chairman: Dr. H. Muller (Cape Town).

4.00 – 4.15 p.m. Dr. B. Kaplan (Cape Town): Diffusion defects following alveolar-capillary block.

4.15 – 4.30 p.m. Drs. S. Zwi, I. W. P. Obel, R. Glyn-Thomas, Mr. D. Roux, Mr. H. I. Goldman, Miss C. Bruyns (Johannesburg): Lung function in kyphoscoliosis before and after corrective surgery.

4.30 – 4.45 p.m. Mr. R. P. Hewitson (Cape Town): Bilateral simultaneous pulmonary resections.

4.45 – 5.00 p.m. Dr. P. M. Baker and Dr. C. N. Barnard (Cape Town): A surgical approach to coronary-artery insufficiency.

Television (Wednesday):

New Science Lecture Theatre.

4.00 – 5.30 p.m. ‚Gastrektomie' — Dr. B. J. van R. Dreyer.

Racing (Wednesday):

1.30 p.m. Race meeting at Durbanville Turf Club (see general information).

WEDNESDAY EVENING

8.15 p.m. Congress Ball, City Hall.

THURSDAY, 28 SEPTEMBER 1961

THURSDAY MORNING

Combined Meeting — Medicine, Surgery, Radiology, Neurosurgery and Neurology (Thursday):

New Science Lecture Theatre.

The Diagnosis and Treatment of Occlusive Arterial Disease.

Chairman: Prof. J. F. Brock (Cape Town).

9.10 – 9.22 a.m. Dr. B. Bronte-Stewart (Cape Town): The pathogenesis of atherosclerosis.

9.22 – 9.34 a.m. Dr. J. M. MacGregor (Cape Town): The diagnosis of carotid-artery occlusion.

9.34 – 9.46 a.m. Dr. P. C. Keet (Cape Town): Cerebral angiography in carotid-artery disease.

9.46 – 9.58 a.m. Prof. J. H. Louw (Cape Town): Surgery of carotid-artery occlusion.

9.58 – 10.10 a.m. Dr. M. B. Denny (Johannesburg): Radiology of peripheral vascular disease.
Tea interval.

11.15 – 11.27 a.m. Dr. J. K. Bremer (Pretoria): Aspects of aortic aneurysms.

11.27 – 11.39 a.m. Dr. L. Stein (Johannesburg): Surgery of aorto-iliac occlusion.

11.39 – 11.51 a.m. Mr. W. G. Schulze (Cape Town): Conservative management of occlusive arterial disease.

11.51 – 12.03 p.m. Dr. M. Suzman (Johannesburg): Medical management of occlusive arterial disease with particular reference to anticoagulants.

Hospital Administration (Thursday):

Department of Architecture, Room B.

Chairman: Dr. J. G. Steyn (East London).

9.10 – 10.45 a.m. Dr. B. M. Burger (Boksburg - Benoni): 'n Pleidooi vir die samesnoering van al ons gesondheidsdienste as een departement.

Chairman: Dr. J. G. Burger (Cape Town).

11.15 a.m. Dr. J. de Beer (Kroonstad, OFS): Sterilisasie operasies.

Paediatrics, Anaesthesia, Medicine (Thursday):

Zoology Lecture Theatre.

Chairman: Prof. O. V. S. Kok (Pretoria).

9.10 – 9.25 a.m. Dr. A. G. Park Ross (Kroonstad, OFS): The practical application of resuscitation in the newborn, with special reference to intermittent positive-pressure respiration.

9.25 – 9.40 a.m. Dr. K. Fismer, Dr. F. C. Friedlander (Pietermaritzburg): Resuscitation in anoxia and asphyxia pallida.

9.40 – 10.05 a.m. Dr. P. M. Smythe (Cape Town): The treatment of tetanus neonatorum with intermittent positive-pressure respiration.

The Experiences with Tetanus at Groote Schuur Hospital over the Past 10 Years.

11.15 – 11.30 a.m. Dr. R. Slome (Cape Town): Incidence, clinical types and results of treatment.

11.30 – 11.45 a.m. Dr. P. M. Smythe (Cape Town): The use of tracheotomy in the treatment of tetanus in the child and adult.

11.45 – 12.00 noon Dr. H. Brown (Cape Town): Analysis of 50 consecutive recent cases of tetanus. The use of intermittent positive-pressure respiration in tetanus.

Obstetrics and Gynaecology (Thursday):

Department of Architecture, Room A.

Chairman: Dr. F. Daubenton (Johannesburg).

9.10 – 9.30 a.m. Prof. J. N. de Villiers en dr. J. P. du Toit (Stellenbosch): Die effekte van die toediening van oksitosiese middels aan die einde van die tweede stadium van kraam.

9.35 – 9.55 a.m. Dr. W. Marais (Cape Town): Colposcopic dissection and histology of normal and abnormal placentae.

10.00 – 10.20 a.m. Dr. J. J. de Wet (Stellenbosch): Analise van placenta praevia by Karl Bremer-hospitaal.

10.25 – 10.45 a.m. Dr. L. Resnick (Cape Town): Cervical pregnancy.

10.50 – 11.15 a.m. Tea interval.

11.15 – 11.35 a.m. Dr. J. P. du Toit (Stellenbosch): The incompetent cervix.

Medicine, Thoracic Surgery, Pathology (Thursday):
Psychology Lecture Theatre.
Symposium on 'Rheumatic Heart Disease':
Chairman: Prof. A. J. Brink (Stellenbosch).

9.10 – 9.25 a.m.	Dr. J. L. Braudo (Johannesburg): Acute rheumatic fever — experience with 100 cases.
9.25 – 9.55 a.m.	Dr. J. C. van der Spuy (Pretoria): The clinical pathology of rheumatic mitral disease.
9.55 – 10.10 a.m.	Messrs. L. A. du Plessis, P. E. Marchand, D. N. Fuller and C. Beckerling (Johannesburg): The mitral valve — an anatomical and experimental study.
10.10 – 10.25 a.m.	Mr. D. N. Fuller, Dr. M. M. Zion and Dr. J. L. Braudo (Johannesburg): Repeat mitral valvotomy. Tea interval.

Chairman: Dr. C. N. Barnard (Cape Town).

11.15 – 11.30 a.m.	Messrs. P. E. Marchand, D. N. Fuller, L. A. du Plessis and C. Beckerling (Johannesburg): The aortic valve — an anatomical and experimental study.
11.30 a.m.	Discussion: Artificial valves and valve surgery.

Anaesthesia (Thursday):
Botany Lecture Theatre.
Chairman: Prof. H. Grant-Whyte (Durban).

11.15 – 11.45 a.m.	Dr. G. Jackson Rees (Liverpool): A system of paediatric anaesthesia.
11.55 – 12.10 p.m.	Dr. H. H. Samson (Johannesburg): Improvements in design of anaesthetic acrylic equipment.

General Practice, Medicine, Psychiatry, Public Health (Thursday):
Chemistry Lecture Theatre.
Chairman: Dr. L. O. Vercueil (Maraisburg, Tvl).

9.10 – 9.25 a.m.	Dr. M. G. Woolff (Port Elizabeth): Chronic alcoholism — its management as a social and industrial medical problem.
9.25 – 9.40 a.m.	Dr. M. Feldman (Johannesburg): Modern medical management of alcoholism.
9.40 – 9.55 a.m.	Dr. A. Strating (Pretoria): Die sosiale aspekte van alkoholisme en die metodes vir die bestryding van die vraagstuk.
9.55 – 10.10 a.m.	Dr. M. de Villiers (Pretoria): The common factor in the causation of psychoneurosis.
10.10 – 10.25 a.m.	Dr. R. M. Schweitzer (East London): The value of handwriting analysis in medical practice.

Dermatology (Thursday):
Groote Schuur Hospital, E-Floor Lecture Theatre.
Chairman: Dr. J. J. Jacobson (Cape Town).

9.00 a.m.	Dr. H. D. Brede (Stellenbosch): Microbiology and dermatology with special reference to some observations on fungus infections in the Cape.
9.30 a.m.	Clinical meeting.
10.45 – 11.15 a.m.	Tea interval.

Group Business Meetings (Thursday):

9.15 a.m.	Ophthalmology — Arts Block, Room C.
9.15 a.m.	Symposium on the work of Sisters Incorporated — Arts Lecture Theatre.

THURSDAY AFTERNOON

12.15 – 12.45 p.m.	Congress photograph.
1.00 p.m.	Luncheon, South African Society of Medical Women, Kirstenbosch Tea Room.

Group Meeting (Thursday):

2.15 p.m.	South African Society of Medical Women — Arts Block, Room B.

Television (Thursday):
New Science Lecture Theatre.

2.00 – 3.30 p.m.	'Vaginal hysterectomy' — Prof. J. T. Louw.
4.00 – 5.30 p.m.	‚Totale abdominale histerektomie' — Prof. J. N. de Villiers. 'Caesarean section and resuscitation of infant' — Drs. P. F. M. du Toit and R. F. Maggs.

Sports (Thursday):
Men's Golf and Bowls Competitions take place on Thursday afternoon (see general information).

THURSDAY EVENING

8.15 p.m.	Congress Banquet, Weizmann Hall, Sea Point.

Alternative Entertainment:

8.15 p.m.	Caltex Show, Zionist Hall, Hope Street, Cape Town.

FRIDAY, 29 SEPTEMBER 1961
FRIDAY MORNING

Thoracic Surgery, Radiology, Pathology, Radiotherapy, Medicine (Friday):
New Science Lecture Theatre.
Chairman: Dr. A. Landau (Cape Town).
Symposium on 'Pleural and Pulmonary Neoplasia':

9.10 – 9.25 a.m.	Mr. E. J. Joubert (Johannesburg): Carcinoma of the lung — some clinical features and the radiological appearance in these cases.
9.25 – 9.40 a.m.	Mr. D. Fuller (Johannesburg): Neuropathies, arthropathies and arthralgias associated with bronchogenic carcinoma.
9.40 – 9.55 a.m.	Dr. C. J. Uys (Cape Town): Carcinoma of the bronchus.
9.55 – 10.10 a.m.	Dr. J. C. Wagner, Dr. C. Sleggs and Mr. P. Marchand (Johannesburg): Mesothelioma of the pleura — clinical and epidemiological study.
10.10 – 10.20 a.m.	Dr. J. C. Wagner (Johannesburg): Pathology of pleural mesotheliomas.
10.20 – 10.30 a.m.	Dr. M. A. de Kock (Stellenbosch): Naaldbiopsies van die pleura. Tea interval.

Chairman: Prof. J. Muir Grieve (Cape Town).

11.15 – 11.55 a.m.	Prof. H. Druckrey (Germany): The combination of surgery and chemotherapy in the treatment of cancer.
11.55 a.m. – 12.10 p.m.	Dr. J. D. Anderson (Cape Town): The management of malignant disease of the pleura by radiotherapy and cytotoxic drugs.
12.10 – 12.20 p.m.	Dr. T. Fichardt (Pretoria): Experience with telecobalt-bomb therapy with special reference to cancer of the bronchus.

Group Business Meetings (Friday):

9.15 a.m.	Hospital administration — Arts Block, Room A.
9.15 a.m.	Dermatology — Arts Block, Room B.
11.15 a.m.	Inaugural Meeting of the Faculty of Dermatologists of the S.A. College of Physicians, Surgeons and Gynaecologists — Arts Block, Room B.
11.15 a.m.	Obstetrics and Gynaecology — Arts Block, Room C.

Obstetrics and Gynaecology (Friday):
Department of Architecture, Room C.
Chairman: Dr. P. Connan (Bloemfontein).

9.10 – 9.30 a.m.	Prof. J. T. Louw (Cape Town): The grosser variations and simple explanations of prolapse.

9.35 – 9.55 a.m. Dr. H. Ross (Cape Town): Labour following caesarean section.
10.00 – 10.20 a.m. Dr. D. P. de Villiers (Cape Town): Genital infection (intra-abdominal) in women.
10.25 – 10.45 a.m. Dr. A. H. Lasbrey (Durban): The study of symptomatic sequelae of symphisiotomy performed on 100 patients.

Surgery (Friday):
Psychology Lecture Theatre.
Chairman: Mr. R. L. Forsyth (Cape Town).
9.15 – 9.35 a.m. Dr. J. K. Bremer (Pretoria): Die tegniek van reseksie van aneurismata van die abdominale aorta.
9.35 – 9.50 a.m. Mr. P. Helman (Cape Town): The treatment of advanced breast cancer.
9.50 – 10.05 a.m. Mr. W. Kark (Johannesburg): The masquerades of acute appendicitis in children and adolescents.

Surgery (Friday):
Department of Architecture, Room B.
Chairman: Mr. W. Kark (Johannesburg).
11.15 – 11.30 a.m. Dr. F. J. Dunai (Windhoek): Assessment of the surgical treatment of tuberculosis of the cervical lymph nodes (film).
11.30 – 11.45 a.m. Mr. H. Katz (Cape Town): A follow-up of 200 cholecystectomy patients with special reference to choledochotomy.
11.45 a.m. – 12 noon Dr. J. J. W. van Zyl (Stellenbosch): Die tegniek van intraveneuse vogtoediening (film).
12.00 – 12.15 p.m. Mr. L. C. J. van Rensburg (Cape Town): Lymphatic perfusion and its application in the treatment of lymph-node pathology.

Ophthalmology (Friday):
Geology Lecture Room.
Chairman: Dr. N. L. Murray (Pretoria).
9.10 – 9.30 a.m. Dr. L. Schrire (Cape Town): One-eyed people in fact and fiction.

FILM SESSION

9.30 – 10.45 a.m. Exenteration of the orbit — Prof. A. B. Reese (New York).
Kronlein operation — Prof. A. B. Reese (New York).
Excision of iris tumours — Prof. A. B. Reese (New York).
Tea interval.
11.15 a.m. – 12.30 p.m. Anterior-chamber implants (sound) 20 mins. — Prof. D. P. Choyce (London).
Anterior-chamber implants (silent) 20 mins. — Prof. D. P. Choyce (London).
Lens implants (silent) 40 mins. Dr. E. Epstein (Johannesburg).
Extracting the cataract (silent) 20 mins. Dr. R. A. Trope (Johannesburg).
(Subject to alteration).
Lunch interval.

General Practice, Medicine, Anaesthesia, Paediatrics (Friday):
Chemistry Building, New Lecture Theatre.
Chairman: Prof. O. V. S. Kok (Pretoria).
Symposium on 'Respiratory Failure':
9.10 – 9.30 a.m. Prof. G. A. Elliott (Johannesburg): The meaning of respiratory failure.
9.30 – 10.00 a.m. Dr. S. Zwi (Johannesburg): The early recognition of respiratory failure, with simple methods of assessing respiratory function.
10.00 – 10.20 a.m. Dr. A. B. Bull (Cape Town): Practical management of respiratory failure.
10.45 – 11.15 a.m. Tea interval.
11.15 – 11.30 a.m. Dr. J. Horak (Pretoria): Fitness for air travel.
11.30 – 11.55 a.m. Dr. G. Jackson Rees (Liverpool): Special considerations of respiratory failure in children.
11.55 a.m. – 12.10 p.m. Dr. J. C. Nicholson (Johannesburg): Post-operative disturbances of pulmonary ventilation.
12.10 – 12.25 p.m. Dr. S. H. Ripley (Durban): Treatment of drowning.

Medicine (Friday):
Arts Block, Large Lecture Theatre.
Chairman: Dr. P. Leftwich (Johannesburg).
9.10 – 9.30 a.m. Dr. Pericles Menof (Johannesburg) Atherosclerosis — its causation and control.

Gastric Function in Ischaemic Heart Disease:
9.35 – 9.50 a.m. Dr. B. Bronte-Stewart (Cape Town): Fat-tolerance test.
9.50 – 10.05 a.m. Drs. S. Bank and I. N. Marks (Cape Town): Acid secretory function.
10.05 – 10.20 a.m. Dr. L. Krut (Cape Town): Gastric lipolytic activity.

Medicine (Friday):
Zoology Lecture Theatre.
Chairman: Dr. N. A. Rossiter (Durban).
9.10 – 9.25 a.m. Drs. L. Vogelpoel, V. Schrire, W. Beck, M. Nellen and A Swanepoel (Cape Town): The atypical systolic murmur of minute ventricular septal defect and its recognition by amyl nitrite and phenylephrine.
9.25 – 9.40 a.m. Dr. J. B. Barlow and Dr. W. A. Pocock (Johannesburg): The significance of aortic systolic murmurs.
9.40 – 9.55 a.m. Dr. V. Schrire and Dr. L. Vogelpoel (Cape Town): The splitting of the second heart sound associated with dilatation of pulmonary arteries.
9.55 – 10.10 a.m. Drs. L. Vogelpoel, V. Schrire, W. Beck, A. Swanepoel and M. Nellen (Cape Town): The paradoxical responses of the murmurs to vasoactive drugs in ventricular septal defect and patent ductus arteriosus with pulmonary hypertension.
10.10 – 10.25 a.m. Drs. W. Beck, V. Schrire, L. Vogelpoel (Cape Town): The haemodynamic effect of phenylephrine and amyl nitrite in patients with severe pulmonary hypertension.
10.45 – 11.15 a.m. Tea interval.
11.15 – 11.30 a.m. Dr. M. M. Zion and Dr. B. A. Bradlow (Johannesburg): Quinidine therapy.
11.30 – 11.45 a.m. Dr. L. Schamroth (Johannesburg): The significance of left axis deviation.
11.45 a.m. – 12 noon Drs. R. Fouche, W. Beck, V. Schrire (Cape Town): The radiological assessment of pulmonary blood flow in secundum atrial defect.
12.00 – 12.15 p.m. Mr. C. Beckerling, Dr. J. B. Barlow and Mr. P. Marchand (Johannesburg): The syndrome of dextroversion, anomalous venous drainage and lobar agenesis.

FRIDAY AFTERNOON

Television (Friday):
New Science Lecture Theatre.
12.30 – 2.00 p.m. Mr. R. L. Forsyth (Cape Town): Cholecystectomy.

Group Business Meetings (Friday):
4.00 p.m. Orthopaedics — Arts Block, Room A.
4.00 p.m. Paediatrics — Arts Block, Room B.
4.00 p.m. Geriatrics — Arts Block, Room C.

Symposium on 'Antibiotics, Chemotherapy and Cross-infection' (Friday):

New Science Lecture Theatre.

Chairman: Dr. D. Ordman (Johannesburg).

2.15 – 2.25 p.m. Prof. A. Kipps (Cape Town): Principles in antibiotic therapy.
2.25 – 2.35 p.m. Dr. D. McKenzie (Cape Town): Chemotherapy in staphylococcal infection.
2.35 – 2.45 p.m. Dr. H. R. Ackermann (Cape Town): Chemotherapy in tuberculous meningitis.
2.45 – 2.55 p.m. Dr. S. Esrachowitz (Cape Town): Chemotherapy in acute meningitis.
2.55 – 3.05 p.m. Prof. L. Eales (Cape Town): Dangers of antibiotic therapy.
Tea interval.

Chemistry Building, New Lecture Theatre.

4.00 p.m. Prof. J. F. Murray, Dr. L. Schrire, Dr. H. J. Koornhof, Prof. D. J. du Plessis and Mr. J. A. Myburgh (Johannesburg): Surgical sepsis in the Johannesburg General Hospital.
Dr. W. du T. Naude (Cape Town): Bacteriophage typing of staphylococci from local hospitals.

Orthopaedics (Friday):

Department of Architecture, Room A.

Chairman: Mr. G. Dommisse (Pretoria).

2.15 – 3.00 p.m. Mr. Johan du Toit (Pretoria): (*i*) Film — anterior spinal fusion; (*ii*) Film — Lapedus procedure for hallux valgus.

Physical Medicine, Industrial Medicine, Orthopaedics, Geriatrics (Friday):

Department of Architecture, Room C.

Chairman: Dr. C. W. Coplans (Cape Town).

2.15 – 3.15 p.m. Dr. A. L. Lomey (Johannesburg): Report on a visit to:
(*a*) The Goodwill Industries of America;
(*b*) Home for aged and infirm Hebrews, New York;
(*c*) Workmen's Compensation Rehabilitation Centre, Toronto, Canada.
Tea interval.
4.00 – 4.30 p.m. Dr. D. I. Guthrie (Cape Town): The difficulties and rewards of establishing an industrial rehabilitation centre.
4.30 – 5.00 p.m. Dr. A. L. Lomey (Johannesburg): A bed-to-job project — the physical medicine rehabilitation clinic, Johannesburg General Hospital.

Note: Visits to Dr. Guthrie's Rehabilitation Centre at the Dynamite Factory, Somerset West, may be arranged by request. All are welcome.

Paediatrics (Friday):

Department of Architecture, Room D.

Chairman: Dr. F. P. Walt (Durban).

2.15 – 2.30 p.m. Dr. J. Rabkin (Cape Town): Reading disability in children — diagnosis and treatment.
2.30 – 2.45 p.m. Dr. S. Katz (Cape Town): Two unusual intestinal conditions: (*a*) inspissated milk syndrome; (*b*) trichomonas diarrhoea.
2.45 – 3.00 p.m. Drs. K. Fismer, F. C. Friedlander (Pietermaritzburg): Congestive cardiac failure in acute nephritis.

Medicine, Surgery, Pathology (Friday):

Zoology Lecture Theatre.

Chairman: Mr. G. Sacks (Cape Town).

The Value of the Augmented Histamine Test. Gastroscopy and Gastric Cytology in the Diagnosis and Management of Gastroduodenal Disease:

2.15 – 2.25 p.m. Dr. I. N. Marks, Dr. S. Bank and Miss B. van Embden (Cape Town): The augmented histamine test.
2.25 – 2.35 p.m. Dr. S. Bank and Dr. I. N. Marks (Cape Town): Gastroscopy.
2.35 – 2.45 p.m. Dr. C. Watson (Cape Town): Gastric exfoliative cytology.
2.45 – 2.55 p.m. Prof. J. H. Louw (Cape Town): Surgical application of these tests.
2.55 – 3.10 p.m. Mr. W. Silber (Cape Town): Interpretation, diagnosis and treatment of heartburn.
Tea interval.
4.00 – 4.15 p.m. Drs. I. N. Marks, S. Bank, C. Watson (Cape Town): The value of the pancreatic-function test in the diagnosis of pancreatic, biliary and duodenal disease.
4.15 – 4.30 p.m. Dr. L. Werbeloff (Cape Town): The earlier X-ray detection of masses in the head of the pancreas.
4.30 – 4.45 p.m. Mr. W. Silber (Cape Town): Aspects of achalasia.

Neurology, Psychiatry and Neuro-surgery (Friday):

Department of Architecture, Room B.

Chairman: Dr. R. W. S. Cheetham (Durban).

2.15 – 2.45 p.m. Dr. P. J. Fischer (Krugersdorp): Problems in Bantu psychiatry.
Tea interval.
4.00 – 4.20 p.m. Dr. W. Kluge (Cape Town): Phenylketonuria in mental defectives (together with demonstration of cases in Wards F1 and F2 at Groote Schuur Hospital).
4.30 p.m. Neurological and neuro-surgical case conference arranged by departments of neurology and neuro-surgery at Groote Schuur Hospital (Wards F1 and F2).

Anaesthesia (Friday):

Department of Architecture, Room E.

Chairman: Dr. P. A. Foster (Stellenbosch).

2.15 – 2.30 p.m. Prof. O. V. S. Kok, Drs. L. S. de Villiers, A. J. Cilliers, O. A. Oosthuizen, T. N. Dreyer (Pretoria): Penthrane (methoxyflurane) — a new inhalation anaesthetic.
2.35 – 2.50 p.m. Dr. J. Ozinsky (Cape Town): Hypothermic fibrillation of the heart.
2.55 – 3.10 p.m. Dr. H. M. Curwen (Durban): Caudal analgesia in the neonate.
Tea interval.

Chairman: Dr. A. B. Bull (Cape Town).

4.00 – 4.15 p.m. Prof. H. Grant-Whyte (Durban): Irrational polypharmacy in anaesthesia.
4.25 – 4.40 p.m. Dr. P. A. Foster (Stellenbosch): The effects of 'deaner' on the neuromuscular junction.
4.50 – 5.05 p.m. Dr. G. J. Rossouw and Dr. J. du Plessis (Cape Town): The oxygen-carrying capacity of stored blood.

Announcement by Chairman, Section of Anaesthesia, Prof. O. V. S. Kok, of winner of Moore-Dyke Registrar's Prize.

Television (Friday):

New Science Lecture Theatre.

4.00 – 5.30 p.m. Dr. J. J. W. van Zyl: Ambulant and operative treatment of varicose veins.

FRIDAY EVENING

8.00 p.m. Khalifa, Woodstock Town Hall.

SATURDAY, 30 SEPTEMBER 1961

1.30 p.m. Race Meeting, South African Turf Club, Kenilworth (see general information).

INTENTION FORM

An Intention Form is printed on page xxxiv. Participants in the above programme are particularly requested to return their forms without delay to the Congress Office, P.O. Box 643, Cape Town.

Jaques Roux, M.R.C.O.G., and G. S. Muller Botha, F.R.C.S.
Hon. Organizing Secretaries

IN DIE VERBYGAAN : PASSING EVENTS

The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 11 September 1961 at 5.10 p.m. in the Institute Lecture Theatre. Dr. B. M. Bloomberg will speak on 'Mellituria'.

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South African Paediatric Association (M.A.S.A.), Cape Town Sub-Group. The next meeting of this Sub-group will be held on Tuesday 5 September 1961 in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, at 8.15 p.m. Dr. J. L. van Selm will speak on 'Common ophthalmological conditions in childhood'. All those interested are welcome to attend this meeting.

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University of Natal, Medical Graduates Association. The Alumni Dinner will be held on Monday 9 October 1961 at the Bolton Hall, Albert Street, Durban, at 8 p.m.

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A Clinico-pathological Conference will be held at 4 p.m. on Thursday 7 September in the Tutorial Room, Pathology Department, Medical School, Observatory, Cape. These conferences are held every second Thursday, alternating with Research Forum.

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Dr. S. Lopis, of Johannesburg, has returned to South Africa from his visit to Endocrine and Diabetic Units overseas.

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Mr. J. F. P. Erasmus, neuro-surgeon, formerly of Cape Town and Pretoria, has joined Mr. M. J. Joubert of Durban in his neuro-surgical practice at 113 Sanlam Building, Smith Street, Durban. Telephone: Rooms 65689, 66311.

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 5 September at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Prof. J. H. Louw will speak on 'Surgery of the carotid artery'.

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Maybaker (S.A.) (Pty.) Ltd. announce that, with effect from 11 September 1961, the address of their Johannesburg branch office will be 111, Smit Street, Braamfontein. P.O. Box 3926. The branch will be closed between 31 August and 11 September for the purpose of the transfer, and during this period orders, etc., should be sent direct to P.O. Box 1130, Port Elizabeth, for attention.

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Mr. A. W. Brookes Heywood, M.B. (Cape Town), F.R.C.S. (Edin.), M.Ch. (Orth.) (L'pool), has commenced practice at 911 Medical Centre, Cape Town. Mr. Heywood, formerly a practitioner at Kokstad, was awarded the Baron Hartley Scholarship in 1956 for surgical studies at Edinburgh and Liverpool, and has been attached to the staff of the Karl Bremer Hospital since his return to South Africa. Telephones: Rooms 35497, residence 693071.

Dr. A. W. Brookes Heywood, M.B. (Kaapstad), F.R.C.S. (Edin.), M.Ch. (Orth.) (L'pool), het begin praktiseer as ortopediese chirurg te 911 Mediese Sentrum, Kaapstad. Dr. Heywood, wat voorheen 'n praktisyn op Kokstad was, het in 1956 die Baron Hartley-beurs ontvang vir verdere studies in chirurgie in Edinburgh en Liverpool. Sedert sy terugkeer na Suid-Afrika was hy op die personeel van die Karl Bremer-hospitaal. Telefoon: Kamers 35497, woning 693071.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

FIORINAL ®

Sandoz Pharmaceutical Products announce the introduction of Fiorinal, and supply the following information:

Description. Fiorinal, specifically designed for pain associated with tension, has been developed by Sandoz Limited, in collaboration with the famous headache clinic at Montefiore Hospital, hence its name. Fiorinal is a non-narcotic analgesic, containing the sedative isobutylallybarbituric acid in addition to the well known combination of aspirin, phenacetin and caffeine. The amount of barbiturate in Fiorinal is sufficiently large to make it 'P.H.D.', thus physicians are assured that patients cannot obtain Fiorinal over the counter, but only on prescription.

Indications. Tension Headache — an evaluation in 1,000 patients over a 7 year period — 'The most effective symptomatic medication in the treatment of tension headache have been several analgesic and sedative combinations. One of the most effective is Fiorinal, which yielded relief in two out of three patients.' *Menstrual Pain.* 'An initial dose of two tablets of Fiorinal, taken at the first sign of "cramps", and followed routinely in three hours by one tablet, proved effective in twenty-two cases. Twenty-five cases required an increased or additional dosage of Fiorinal. Thirty-five of forty-seven showed excellent response. No side effects were noted in any of the forty-seven cases'.

Composition. Isobutylallylbarbituric acid 50 mg. (¾ gr.) Acetylsalicylic acid 200 mg. (3 gr.) Phenacetin 130 mg. (2 gr.) Caffeine 40 mg. (¾ gr.)

Presentation. Tubes of 25 and bottles of 250 tablets.

Samples and literature available from Sandoz Pharmaceutical Department, Alex. Lipworth Limited, P.O. Box 4461, Johannesburg.

NORLUTIN-A

Parke, Davis announce the introduction of NORLUTIN-A, a progesterone which exerts a powerful effect when given by the oral route, and supply the following information:

Description. Norlutin-A is the acetic acid ester of norethisterone (17 - acetoxy-17-ethynyl-oestr-4-4n-3-one). It provides potent, orally effective progestational action. According to studies by McGinty in animals, and clinical investigation studies by Greenblatt, Kupperman and others, norethisterone acetate is twice as potent as norethisterone.

Indications. The utility of Norlutin-A has been established in a variety of gynaecological disorders including amenorrhoea, menstrual irregularities, functional uterine bleeding, endocrine infertility, habitual abortion, threatened abortion, premenstrual tension, dysmenorrhoea, and endometriosis. It may also be used as a test for pregnancy. NORLUTIN-A possesses an inherent oestrogenic activity and, therefore, oestrogen priming is usually unnecessary.

Dosage and Administration. Therapy with Norlutin-A should be adapted to the specific indication and to the therapeutic response of each patient as set out in the literature available from Parke-Davis.

Side-effects. Varying degrees of masculinization of the female foetus with the administration of progesterone or progesterone-like compounds, including norethisterone, to gravidas have been reported. Other untoward effects have not been a problem with Norlutin-A. Spotting before the calculated onset, or after termination of menstruation, transient lethargy and nausea, hirsutism, and acne have been reported, but have not ordinarily caused significant problems.

Presentation. Norlutin-A is supplied in bottles of 15×2·5 mg. tablets.

Further information may be obtained from Parke, Davis Laboratories (Pty.) Ltd., P.O. Box 24, Isando, Tvl.

BOEKBESPREKINGS : BOOK REVIEWS

PARKINSONISM

Parkinsonism. Its Medical and Surgical Therapy. By Irving S. Cooper, M.D., Ph.D., F.A.C.S. Pp. xii + 239. Illustrated. R13.20. Oxford: Blackwell Scientific Publications and Springfield, Illinois: Charles C. Thomas. 1961.

This monograph describes the various aspects of parkinsonism. The well-known syndrome is explained and important variations are discussed, such as the akinetic variety. A pithy description of psychological disturbances occurring in this condition is very welcome. Medical therapy is discussed in some detail and some useful hints included whereby medicines can be combined, increased, or temporarily suspended for optimal effect.

The development of surgical therapy makes interesting reading and each step is briefly described with the author's comments on the procedures. The main section is a detailed description of the Cooper technique of chemopallidotomy and thalamotomy, the histories of descriptive cases and the results obtained in 1,500 consecutive operations.

The author concludes that the surgical target of choice is the region of the ventro-lateral nucleus of the thalamus. A suitable lesion here will relieve rigidity and tremor in 90% of subjects who are suitably selected.

This is a well-planned and beautifully produced book that will be of interest to all dealing with extrapyramidal disorders. A.G.

DRUG THERAPY

The Year Book of Drug Therapy — 1960-61. Ed. by Harry Beckman, M.D. Pp. 587. 57 illustrations. $8.50. Chicago: The Year Book Publishers. 1961.

This useful volume, which makes its welcome appearance annually, has all the good features of its predecessors. It is handy in size, and is filled with abstracts from a great variety of journals, pithy editorial comments not always favourable to the drug investigated, and a double index of authors and subjects.

This edition, like that of last year, is virtually two books in one. In addition to the traditional abstracts, grouped according to the main disorders for which drugs are used, there is, on blue paper, a separate section of some 60 pages in which the year's new drugs are critically considered in concise manner. More than 200 preparations are reviewed, and this does not include consideration of multiple-vitamin preparations and new dosage forms. Fortunately most of these preparations and their names, have not reached this country, but the general remarks and criticisms are important.

This book is certainly worth getting; there is something of interest for all members of the medical profession. N.S.

UROLOGY

The Year Book of Urology (1960-1961 *Year Book Series*). Ed. by William Wallace Scott, M.D., Ph.D. Pp. 400. 79 illustrations. $8.00. Chicago: Year Book Medical Publishers. 1961.

Year Books are always fascinating and, more often than not, stimulating. This volume incorporates, for the first time, editorial associates who are recognized experts in some particular field of urology and who write introductory notes and comments in their special sections. While this innovation seemingly adds erudition, there is a danger that in time editorial comment may occupy an overlarge and quite unwarranted amount of space. Basically Year Books collect articles of interest for perusal by specialists in a particular subject: there is little need for the introduction of a number of critics.

Once again the selection is wide and variable. Why certain articles are included and others ignored continues to remain a yearly mystery to all except the Editor; the complete absence of a single abstract from the *British Journal of Urology* is still more mysterious. I.J.

OPHTHALMOLOGY

Year Book of Ophthalmology. (1960-1961 Year Book Series) Edited by William F. Hughes, M.D. Pp. 375. Illustrated. $8.50. Chicago: The Year Book Medical Publishers. 1961.

The publication of the latest Year Book demonstrates once more the popularity of this type of survey of the world literature, its brevity being useful for the busy practitioner and a reminder of earlier reading. The printing and presentation of material is again of the highest standard in this second volume under the editorship of William F. Hughes, who himself supplies the special article on the management of uveitis. The remainder of the volume follows the pattern of previous years in having fairly lengthy abstracts, with critical editorial comment, on the most significant articles that have appeared between September 1959 and August 1960. The commentary on certain subjects has been delegated to qualified experts, whose names are appended, but has been rather less than before. This is regrettable, since the commentary is often as valuable as the article itself. This volume, like its predecessors, therefore, is a convenient method of keeping up with a literature which is steadily increasing in volume. L.S.

ELEKTRON-MIKROSKOPIE

Electron Microscopy in Anatomy. Proceedings of a Symposium held by the Anatomical Society of Great Britain on the Ultra-structure of Cells. Pp. viii + 288. Illustrated. R5.00. London: Edward Arnold Publishers. 1961.

Hierdie simposium bestaan uit twintig artikels deur verskillende navorsers. Die eerste 5 artikels handel oor tegniese probleme soos die evaluasie van die moontlikhede van ultramikroskopie, bevriesingsmetodes van fiksering, die maak van koolstofreplikas van weefsel wat normaalweg nie met die elektron-mikroskoop ondersoek kan word nie, e.s.m. Die vooruitsig vir 'n kleiner en goedkoper elektron-mikroskoop, wat die aanskaffing van so 'n instrument vir baie inrigtings sal moontlik maak, word daargestel.

Die ander 15 artikels handel oor die ultrastruktuur van selle, membrane, sinapse, neuromuskulêre aansluitings, en senuweevesels. Die miëliniseringsproses van perifere senuwees, sowel as die verhouding van die Schwann-selle tot perifere senuweevesels, word baie goed uitgebeeld. Dit bevat belangrike en interessante leesstof vir die anatoom en die histoloog in die besonder en vir die mediese leser in die algemeen.

Die elektron-mikrografiese reproduksies is besonder duidelik afgedruk met veelvuldige wysers wat die verwysing in die teks na die afdrukke baie vergemaklik. J.F.v.E.K.

BRIEWERUBRIEK : CORRESPONDENCE

HEARTBURN IN PREGNANCY

To the Editor: Dr. Quinlan's article[1] in the *Journal* of 29 July on heartburn in pregnancy was most interesting.

Maybe he could offer some explanation for the gratifying results I have been getting with Co-pyronil caps. (an antihistaminic preparation) 1 b.i.d. for heartburn in pregnancy. Please note that these capsules work only in those patients who get no relief with alkalis.

My original idea was that the heartburn was caused by an allergic factor. I have had to drop that theory because I later used less expensive long-acting antihistaminics, which gave good results in other allergies, but they gave no results in my heartburn cases.

M. J. Goddefroy

Medfontein
Bloemfontein
10 August 1961

1. Quinlan, D. K. (1961): S. Afr. Med. J., 35, 628.